EXHIBIT 11

UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF NEW YORK

IN RE: Acetaminophen – ASD-ADHD Products Liability Litigation **This Document Relates To:** *All Cases*	Docket No. 22-md-3043 (DLC)

AMENDED RULE 26 EXPERT REPORT OF DR. ERIC HOLLANDER, M.D., DFAPA, FACNP

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I. EXECUTIVE SUMMARY

The following amended report is provided pursuant to Rule 26 of the Federal Rules of Civil Procedure. The materials that I have considered in forming my opinions are included in this report and within my materials considered list. I have also reviewed the expert reports of Dr. Andrea Baccarelli, M.D., PhD (epidemiology), Dr. Robert Cabrera, PhD (teratology and genetics), Dr. Brandon Pearson, MSc, PhD (toxicology), and Dr. Stan Louie, PharmD (pharmacology), and all materials considered cited therein. I have based my opinions on my background, education, experience, and knowledge, my own review of the medical and scientific literature, as well as the materials provided to me.

I am a licensed psychiatrist with expertise in psychopharmacology and neuropsychopharmacology. I have been asked to review the medical and scientific literature and to apply my professional experience and judgment to opine about the interconnectedness of various neurodevelopmental disorders, including Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD); whether the scientific evidence regarding the association between prenatal exposure to acetaminophen ("APAP") and neurodevelopmental disorders informs the question of whether prenatal exposure to APAP can cause the neurodevelopmental disorders of ASD and ADHD; and to assess whether there are plausible biological mechanisms to explain how APAP can cause the neurodevelopmental disorders of ASD and ADHD. To support my analysis and opinion, I provide an overview of the following: diagnostic criteria for ASD and ADHD, including DSM-IV, DSM-IV-TR, and DSM-5 diagnostic criteria and ICD classifications; the role of genetics and environmental factors in neurodevelopmental disorders; the overlap between ASD and ADHD established through a transdiagnostic approach; pathophysiology of

neurodevelopmental disorders; and neuropsychopharmacological analysis of prenatal exposure to APAP and pharmacological effects on neurodevelopment.

ASD and ADHD are highly heterogenous both in terms of etiology and presentation. In other words, there is no single cause or risk factor, and the disorders manifest in a wide variety of symptoms that have significant, lifelong impacts on individuals, their families, and society as a whole. Because of the variability in ASD and ADHD, treating and researching these conditions is highly complex and requires expertise beyond the traditional boundaries of psychiatric and psychological specialties. Thus, transdiagnostic approaches provide valuable insight.

The transdiagnostic process refers to a mechanism that underlies and connects a group of disorders that transcends traditional diagnostic boundaries. For example, recent transdiagnostic research shows that neurodevelopmental disorders including ASD and ADHD share important neural, genetic, physiological, structural, and psychological traits (Barch, 2020). Both disorders are known to overlap with each other and other neurodevelopmental, psychiatric, and medical disorders. A recent functional magnetic resonance imaging (fMRI) study supports the idea that certain traits of neurodevelopmental disorders transcend traditional diagnostic categories. That study compared images of the brains of children diagnosed with ASD, ADHD, or OCD with those of neurotypical children and identified brain-based subgroups with similar biology among the children with ASD, ADHD, or OCD (Vandewouw et al., 2023). This result was consistent with observed symptomatic commonalities among the disorders indicating alterations in the brain's resting-state functional network (Vandewouw et al., 2023).

Based on my experience and expertise, when analysing causal associations between a toxic exposure and neurodevelopmental disorders such as ASD and ADHD, it is appropriate to consider comprehensive evidence that examines a variety of neurodevelopmental symptoms that could be

associated with ASD and ADHD, and to not limit the review to studies and analyses that solely assess ASD and ADHD diagnoses. Across neurodevelopmental disorders, there is often overlap of symptoms, and neurodevelopmental disorders frequently co-occur. This is true regarding the specific analysis of whether there is a causal association between prenatal exposure to APAP and the neurodevelopmental disorders of ASD and ADHD. As a medical doctor who has treated and researched ASD and ADHD using a holistic, transdiagnostic approach, I have reviewed extensive medical literature from various specialties that assess the impact of prenatal APAP exposure on neurodevelopment. As a researcher and clinician, the volume and breadth of these studies with consistent findings, is notable. The entirety of that literature, as effectively summarized and analyzed in the other expert reports submitted in this case, is relevant to the question of whether APAP can cause the neurodevelopmental disorders of ASD and ADHD. In fact, Johnson & Johnson reviewed broad neurodevelopment studies when assessing the causal association between prenatal use of APAP and the neurodevelopmental disorders of ASD and ADHD (Exhibit 63 to Deposition of [redacted]; Exhibit 80 to Deposition of [redacted]).

In addition, I reviewed the scientific evidence to assess whether there was a plausible biological mechanism by which prenatal use of APAP could impact fetal brain development and cause neurodevelopmental disorders. To address this issue, I provide a background on APAP and how I have recently discovered that APAP's profile as a safe drug for pregnant women may not be as accurate as has been represented to clinicians, like myself, and to the public. In fact, the label for Ultracet, which is a combination of APAP and another drug, tramadol, and is manufactured by a subsidiary of Johnson & Johnson, specifically warns of a fetotoxic animal study as it relates to fetuses. In addition, I outline several, plausible biological mechanisms that are informed by my research and review of the scientific evidence.

Based on my review of this scientific evidence, along with my education, training, decades of clinical and research experience, and a review of information from regulators and manufacturers, I state the following opinions:

- There is a strong interrelationship between the neurodevelopmental disorders of ASD and ADHD and across neurodevelopmental disorders.
- Based on this interconnectedness of neurodevelopmental disorders, including ADHD and ASD, it is appropriate to review the body of evidence that measures symptoms of neurodevelopmental disorders and to not limit the analysis to studies that focus on ASD and ADHD as specified outcomes when evaluating the potential causal association between prenatal APAP exposure and ASD and ADHD in offspring.
- I reviewed the underlying epidemiological literature regarding whether prenatal use of APAP can cause the neurodevelopmental disorders of ASD and ADHD, and that body of literature is consistent with my opinion that to fully investigate and address the question of whether prenatal use of APAP can cause the neurodevelopmental disorders of ASD and ADHD, scientists should look to the full body of neurodevelopmental studies.
- There are multiple, plausible mechanisms of action to explain how APAP can impact fetal brain development and lead to neurodevelopmental disorders in offspring.
- Depending on the timing and duration of APAP exposure to the developing brain, if the suspect mechanisms of injury occur, then one would expect a wide variety of diffuse neurologic symptoms/injuries when the brain develops.

- Based on my review of the literature, the expert reports, and my decades of clinical experience, I would not advise my pregnant patients to take APAP unless they have fever and to use at the lowest effective dose for the shortest time possible and the lowest possible frequency.

I express the opinions set forth herein to a reasonable degree of medical and scientific certainty. In reaching these conclusions, I have followed the same or similar procedures that I employ in my clinical practice and in the clinical study of neurodevelopmental disorders, including ASD and ADHD. I have not used special or different procedures in preparing this report.

II. QUALIFICATIONS AND EXPERIENCE

In my work and career, I primarily have specialized in the field of psychiatric medicine, neurodevelopmental disorders, and neuropsychopharmacology.

I am a psychiatrist licensed to practice medicine in the state of New York. I was board certified in 1987 by the American Board of Psychiatry and Neurology. I am a Professor of Psychiatry and Behavioral Sciences, and the Director of the Autism and Obsessive-Compulsive Spectrum Program, at the Psychiatry Research Institute of Montefiore-Einstein at Albert Einstein College of Medicine (AECOM) and Montefiore Medicine, in the Bronx, New York. I am a fellow of the American College of Neuropsychopharmacology (ACNP), and a Distinguished Lifetime Fellow of the American Psychiatric Association (APA). I am the past president of the International Society of Research in Impulsivity (ISRI).

I have an extensive background in psychopharmacology and neuropsychopharmacology. Neuropsychopharmacology combines neuroscience with pharmacology and studies the effects of drugs on neural mechanisms that influence behavior. I completed a Fellowship in Psychopharmacology at Columbia University and was the Principal Investigator for the National Institute of Mental Health (NIMH) Psychopharmacology Training Program.

Prior to 2009, I was an Esther and Joseph Klingenstein Professor of Psychiatry and Chairman of Psychiatry at Mount Sinai School of Medicine. I was also an attending psychiatrist at Mount Sinai Hospital from 1993 to 2008. From 1986 to 1993, I was faculty at Columbia University College of Physicians and Surgeons and the New York State Psychiatric Institute, and Associate Professor of Clinical Psychiatry.

I received my Medical Degree in 1982 from the State University of New York, Downstate Medical College, Brooklyn, New York. I served my Internship in Internal Medicine at Mount Sinai Hospital in New York, New York, from 1982 to 1983. I was a Resident in Psychiatry at Mount Sinai from 1983 to 1986, serving as Chief Resident–Psychiatry Research from 1985 to 1986. From 1986 to 1988, I was a NIMH Psychiatry Research Fellow at Columbia University College of Physicians and Surgeons. I was a Dana Foundation Fellow at Columbia from 1988 to 1989, and practiced as a NIMH Research Scientist Development Award recipient there from 1988 to 1993.

I have received numerous awards and honors throughout my career, including Best Doctors in New York and Best Doctors in the United States from 2007 until present, and a National Alliance for Research on Schizophrenia & Depression (NARSAD) distinguished investigator award in 2009. I am an inventor on several patents related to, among other things, oxytocin or memantine in autism and other disorders.

I have published more than 500 peer-reviewed scientific papers in professional literature sources. I have edited 20 books, including the Textbook of Autism Spectrum Disorders, 2nd edition (American Psychiatric Publishing, 2022), Textbook of Autism Spectrum Disorders, 1st edition (American Psychiatric Publishing, 2011), three editions of the Textbook of Anxiety, Trauma, and OCD Related Disorders (American Psychiatric Publishing, 2002, 2009, 2020), and the Clinical Manual of Impulse Control Disorders (2006).

I am the Chair of the Board of Directors of the International College of Obsessive Compulsive Spectrum Disorders (ICOCS). The ICOCS is primarily aimed at advancing, promoting and facilitating research into the causes and consequences of obsessive compulsive disorder (OCD) and obsessive compulsive spectrum disorders. The organization intends to aid and stimulate mental health professionals and others to develop research projects in the field, and to help coordinate research efforts amongst members. The organization's website[1] is dedicated to news, research findings, funding opportunities and new developments in the study of OCD and OC spectrum disorders. The organization holds scientific meetings annually for ICOCS members and other interested clinicians and researchers. The ICOCS also increases public health awareness of OCD and OC spectrum disorders with the hope of improving diagnosis and encouraging better deployment of resources for assessment and treatment. Its members offer advice to legislative bodies and government agencies and cooperate on national and international treatment guidelines.

From 2002 to 2007, I served the APA as the Chair of the Diagnostic and Statistical Manual of Mental Disorders ("DSM-5") Research Planning Agenda for Obsessive Compulsive Behavior Spectrum Disorders, and then from 2007 to 2009 as a member of the DSM-5 Anxiety, Obsessive-Compulsive Spectrum, Post-Traumatic and Dissociative Disorders Workgroup, and the Behavioral and Substance Addictions Workgroup. In 1998, I served as the Workgroup Chair for the Impulse Control Disorder Section of the DSM-IV-TR. From 1992 to 1994, I served as a member of the Obsessive-Compulsive Disorder Workgroup for the DSM-IV. In 2006, I edited the APA Publishing Clinical Manual for Impulse Control Disorders. I was also a member of the editorial board of the

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[1] http://www.icocs.org.

APA Treatment Manual for Obsessive Compulsive Disorders, and the 2023 CANMAT/ICOCS Practice Guidelines for Obsessive Compulsive and Related Disorders.

I have served as the principal investigator on a number of federal grant funded research projects. I have received orphan drug grants from the Food and Drug Administration to study new treatments for Prader Willi Syndrome (a neurodevelopmental disorder and syndromic form of ASD), body dysmorphic disorder, child/adolescent autism, and adult autism, and a grant from the National Institute of Drug Abuse for a study on the neurobiology of pathological gambling. I have received several grants from NIMH and the National Institute of Neurological Disorders and Stroke to develop treatments for borderline personality disorder, adolescent body dysmorphic disorder, and autism. I was the principal investigator of the Autism Clinical Trials Network and Chair of the eight centers participating in the National Institutes of Health Studies to Advance Autism Research and Treatment (NIH STAART) Autism Steering Committee. I am involved in research funded by federal institutes, not-for-profit foundations, and by industry on the neuropharmacology, neuropsychiatry, functional imaging, and treatment of multiple disorders including, but not limited to, autism spectrum disorder, obsessive-compulsive disorder, impulsive/aggressive personality disorders, and obsessive-compulsive-related disorders, such as body dysmorphic disorder, attention deficit hyperactivity disorders, impulse control disorders, problematic use of the internet, and pathological gambling. I have served as Principal Investigator for numerous federal grants in Autism Spectrum Disorders, including active grants with NIH STAART studies, DOD CDMRP Autism Research Program, Foundation for Prader Willi Research, Roche Pharmaceuticals, and FDA Orphan Products Division grants. I have previously served as Principal Investigator for federal grants including the NIH Greater New York Autism Center of Excellence, the NIMH Research Training Grant in Psychopharmacology and Outcomes Research,

and the Autism Clinical Trials Network. I have received funding from the Department of Defense for the study of cannabidivarin (CBDV) in ASD, and from the Orphan Products Division of the FDA for the study of intranasal oxytocin in Prader-Willi Syndrome.

I have over 30 years of clinical and translational research experience. I currently serve as the Director of Spectrum Neuroscience and Treatment Institute (SNTI). SNTI offers state of the art, interdisciplinary and personalized integrated treatments for obsessive-compulsive disorders (OCD), anxiety disorders, autism spectrum disorders, and attention-deficit/hyperactivity disorders.

I have used my clinical experience and training to treat people with ASD, ADHD, OCD and related conditions, in addition to Prader-Willi Syndrome, depression, body dysmorphic disorder, anxiety disorders (including Generalized Anxiety Disorder, Social Anxiety and Panic Disorder), depersonalization disorder, cataplexy, trichotillomania, sleep disorders and pain disorders.

In my research and clinical work, I routinely make assessments of potential causal associations between a substance and particular pharmacological effect. As a practitioner who specializes in neuropsychopharmacology, I have undertaken these types of assessments for more than two decades. As a clinician and researcher, my work involves diagnosing neurodevelopmental disorders, including ASD and ADHD, as well as understanding and examining the underlying causes and mechanisms of those disorders. My education, training, and decades of experience in this field qualifies me to render the opinions included in this report.

As a psychiatrist, I have extensive experience diagnosing and treating neurodevelopmental disorders. The experience has led me to identify commonalities among many neurodevelopmental disorders based on the symptoms I evaluate, the assessments and diagnostic tools I use, and the clinical and pharmacological interventions I recommend. While ASD and ADHD in particular are

highly heterogenous, my experience has shown they are likely to overlap symptomatically or be comorbid with each other or with other neurodevelopmental disorders. The scientific research on this topic confirms my experience.

Further, my own clinical and translational research in neuropsychopharmacology enhances my clinical experience by allowing me to assess and draw conclusions about the effects of drugs (like APAP) on neurodevelopmental disorders, including ASD and ADHD. I have decades of experience understanding and examining the underlying causes and mechanisms of neurodevelopmental disorders. In this report, I apply my extensive experience and knowledge to arrive at the opinions stated above.

III. OVERVIEW OF NEURODEVELOPMENTAL DISORDERS, INCLUDING ASD AND ADHD

In my practice as a psychiatrist with a focus in neuropsychopharmacology, I routinely assess neurodevelopmental causation issues in my patients. This includes reviewing, analyzing, conducting, and collaborating with colleagues on clinical and epidemiological studies as both a clinician and clinical researcher. Epidemiological studies, which are derived from observational data experienced by many patients, support my understanding of psychiatry, pharmacology, and neurodevelopment especially at it pertains to causation issues. My expertise and experience allow me to understand and evaluate the epidemiological studies that assess the association between prenatal use of APAP and neurodevelopmental disorders like ASD and ADHD.

ASD and ADHD are permanent neurodevelopmental disorders that are primarily associated with the functioning of the neurological system and the brain. Neurodevelopmental disorders are characterized by developmental deficits that impair personal, social, academic, or occupational functioning (DSM-V). Children with neurodevelopmental disorders can experience difficulties with language and speech, motor skills, behavior, memory, learning and other functions.

Abundant research across decades has shown that children with better attention and executive function do better in every aspect of life, not only in school, but also regarding income earning potential and adult physical health (Liew et al., 2016; Christakis et al., 2016; Moffitt et al., 2011). Both ASD and ADHD can have significant detrimental life impact on the afflicted child and their families in adjusting and supporting such neurodevelopmental disorders, including significant detrimental impact on learning, on social and communication abilities, and on planning, organizing and coping with life challenges, among other facets of life, even for those with above average intelligence.

A. Transdiagnostic Approaches to Developmental Disorders

Although traditional psychiatric and psychological approaches involve focusing on a specific diagnostic category, recent work in the field has demonstrated that these artificial boundaries do not always reflect the constellation of symptoms I see in my patients. The biological factors behind these symptoms cut across traditional diagnostic boundaries, as demonstrated by recent transdiagnostic research that shows shared neural, genetic, physiological, structural, and psychological traits (Barch, 2020). Transdiagnostic is defined as a proposed mechanism that underlies and connects a group of disorders. This process has been the focus of newer research, with the NIMH even creating a Research Domain Criteria (RDOC) Initiative to help provide an organizational structure that considers mental health, neurodevelopment, and psychopathology in the context of major domains of neurobehavioral functioning, rather than within traditional diagnostic categories (*About RDoC*, n.d.).

Both ASD and ADHD are known to overlap and/or be comorbid with each other, in addition to many other neurodevelopmental, psychiatric, and medical disorders. For example, in Vandewouw et al. (2023), researchers compared functional magnetic resonance imaging (fMRI) of the brains of children diagnosed with ASD, ADHD, or OCD to those of children who were

typically developing. They identified brain-based subgroups with similar biology overall in the children with the neurodevelopmental disorders compared to the healthy control children. These brain-based subgroups did not align with current diagnostic categories (Vandewouw et al., 2023). Hyperactivity, externalizing behaviors, conduct problems, emotion regulation difficulties, and impulsivity are observed to be shared characteristics across neurodevelopmental conditions and were associated with alterations in the brain's resting-state functional network, including widespread increased segregation and patterns of both increased and decreased integration (Vandewouw et al., 2023). As a result of both this and other studies, a holistic and transdiagnostic approach that uses continuous measures of behavior is necessary to fully understand the highly heterogenous conditions of ASD and ADHD. Likely for this reason, Johnson & Johnson followed this approach and reviewed broad neurodevelopment studies when assessing the causal association between prenatal use of APAP and the neurodevelopmental disorders of ASD and ADHD (Exhibit 63 to Deposition of [redacted]; Exhibit 80 to Deposition of [redacted]).

B. Autism Spectrum Disorder

B.1. DSM and ICD Diagnostic Criteria and Classification

ASD is a neurodevelopmental disorder characterized by persistent deficits in social communication and social interaction, and the presence of restricted and repetitive patterns of behavior, interests, and activities. There is an estimated 2.8% worldwide prevalence rate of ASD, with the CDC reporting that 1 in 36 children have an ASD diagnosis (Maenner et al., 2020). ASD occurs in all racial, ethnic, and socioeconomic groups and is reported to be nearly four times more common among males than among females. However, this gender gap continues to narrow, and ASD is often underdiagnosed in females. Due to the heterogeneity of ASD, the presentation of symptoms varies across individuals and may be similar to, or overlap with, other neurodevelopmental and psychiatric disorders.

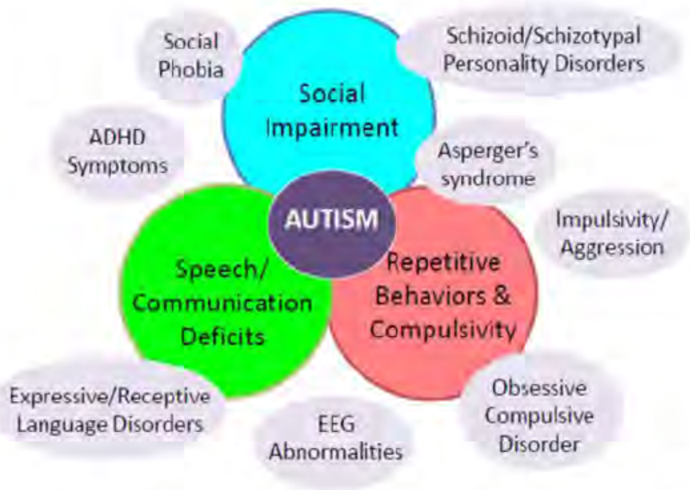
The DSM-IV was published in 1994, with a revised version released in 2000 (DSM-IV-TR). The current version of the DSM is the DSM-5, which was published in 2013. As the studies discussed in this report include individuals diagnosed under the diagnostic criteria set forth in either the DSM-IV or DSM-5, I discuss the diagnostic criteria set forth in each edition. The complete DSM-5 and DSM-IV diagnostic criteria for autistic disorder and ASD are excerpted in **Appendix 1.**

Several important changes relating to ASD and ADHD were made to the DSM-5 compared to the DSM-IV-TR (Doernberg & Hollander, 2016). Notably, the chapter of the DSM-IV-TR titled "Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence"—which included autism and a host of other conditions—was deleted and replaced with a chapter titled "Neurodevelopmental Disorders." Regarding autism in particular, the DSM-5 removed the five separate pervasive developmental disorders (autistic disorder, Asperger's disorder, Rett's disorder, childhood disintegrative disorder, and PDD Not Otherwise Specified, or NOS) and created the "spectrum" diagnosis, reducing autism to two core domains (DSM-IV-TR; Doernberg & Hollander, 2016).

As noted above, in the DSM-IV-TR, the autism spectrum disorder diagnosis was not singular. Instead, three different diagnoses existed separately: Autistic Disorder (299.0), Asperger's Disorder (299.80), and Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS), including atypical autism (299.80). The Autistic Disorder diagnosis was generally given to those who presented with more severe forms of autism, while the PDD-NOS diagnosis was for those with less severe forms. Asperger's Disorder was generally given to those with higher functioning autism without a history of speech and cognitive delays.

The ICD is a globally-accepted framework for medical diagnosis (World Health Organization, *International Statistical Classification of Diseases and Related Health Problems (ICD)*). Developed and maintained by the WHO, its primary purpose is classification, in contrast to the DSM, which focuses primarily on diagnosis (Doernberg & Hollander, 2016). The ICD has four distinct purposes: facilitating clinical utility by establishing a shared diagnostic language, addressing insurance-related requirements, addressing legal considerations, and ensuring long-term support for research endeavors (Doernberg & Hollander, 2016). As a comprehensive medical system, the ICD encompasses specifiers for identifying underlying causes and mirrors the classification approach used for all other medical conditions (Doernberg & Hollander, 2016). Many of the epidemiological studies that assess the causal association between prenatal use of APAP and neurodevelopmental disorders use the ICD-9 and ICD-10 classifications to identify diagnoses. The complete list of elements of the ICD-9 and ICD-10 are excerpted in **Appendix 1.**

A final issue relating to defining and classifying ASD is use of the term "regressive autism" in some older literature. This is a descriptor previously used to describe children who appeared to meet all developmental milestones until approximately age 2, but then suddenly have a rapid loss of skills. In recent years, this term has lost favor, and top minds in the field, including Sally Ozonoff and Catherine Lord, have confirmed that one cannot split individuals with autism into the camps of regressive or non-regressive. Instead, it is well-recognized that each child presents with differing signs of ASD early in life, and there are a variety of onsets. This diagnostic term is no longer used in the primary literature (*Rethinking Regression in Autism*, 2017).



Core Symptom Domains and Associated Features of Autism Spectrum Disorder

B.2. Sex Differences in ASD Diagnoses.

ASD is reported to be nearly four times more common among males than among females, but some evidence shows the gap is narrowing. The difference in diagnostic rate is also apparent in some research relating to ASD presentation and symptoms. Some research shows no sex differences in restricted and repetitive behaviors (RRBs) for males and females with ASD, while other studies show that RRBs present more subtly in females with ASD (Jamison et al., 2017). Females with ASD are shown to present with more socially acceptable restricted interests and greater play and imaginative skills which may mask unusual behaviors, particularly during childhood (DaWalt et al., 2020; Jamison et al., 2017).

Similar to rates in typically-developing adolescents and adults, females with ASD are at a greater risk for depression and anxiety and present with more internalizing conditions compared to both typically-developing males and males with ASD (DaWalt et al., 2020; Jamison et al., 2017). Additionally, possibly due to implicit biases, females need to present with more severe disruptive behaviors than males in order to receive a diagnosis of ASD. Females socialized to internalize

emotions and behaviors may have better adaptive skills, allowing them to suppress symptoms and fit in until social pressures become too great (Jamison et al., 2017). As a result, depression and anxiety may initially appear as the primary diagnosis during the challenging adolescent developmental period or adulthood (DaWalt et al., 2020; Jamison et al., 2017). Thus, diagnosis of ASD may be late or nonexistent in females.

Research aimed at understanding how sex differences influence social and communication function in these disorders is limited and results are conflicting. Mahendiran et al. investigated potential sex differences across age in social adaptive function in ASD and ADHD compared to typically developing controls (Mahendiran et al., 2019). Participants included 115 youth with ASD, 172 youth with ADHD, and 63 typically developing controls, aged 7 to 13 years, with 75% males. Social adaptive function was assessed using the Adaptive Behavior Assessment System-Second Edition (ABAS-II). Proportions of adaptive behaviors in each skill area were analyzed as binomial outcomes through logistic regression, while accounting for age and investigating age-by-sex interactions. An additional analysis explored the impact of controlling for core symptom severity on the observed sex effect.

In the ASD group, Mahendiran et al. observed significant interactions between sex and age in the areas of communication ($p = 0.005$), leisure ($p = 0.003$), and social skills ($p < 0.0001$) (Mahendiran et al., 2019). In these areas, females scored lower (indicating poorer function) compared to males at older ages, despite females performing better at younger ages. Significant sex-by-age interactions were also found between ASD and typically developing controls in the social and leisure domains. Typically developing females showed better scores at older ages compared to younger ages. These findings highlight the importance of considering a

developmental perspective when examining sex differences, which may have implications for diagnosis, prognosis, and treatment.

B.3. Associated Features and Co-morbid Psychiatric and Medical Disorders

Individuals with ASD often present for treatment for symptoms that fall outside of the core symptom domains, including aggression, irritability and self-injurious behaviors. Approximately 70% of individuals with ASD have a co-morbid mental health diagnosis, and 40% have two or more co-morbid mental health diagnoses. ADHD is the most frequent comorbid disorder with ASD.

Psychiatric comorbidities are common in ASD and are associated with increased symptom severity across the lifespan. Anxiety and depression are frequently associated with ASD (Gotham et al., 2015; Towbin et al., 2005). As compared to the general population, those with ASD have significantly higher rates of suicide, with one surveyed sample noting that 66% reported suicidal ideation (Cassidy et al., 2014). Bipolar disorder and ASD often overlap as they share common etiological factors (Sullivan et al., 2012). Individuals with ASD and comorbid OCD may present with unique behavioral profiles and tend to exhibit higher self-reported obsessive-compulsive symptoms than individuals diagnosed with only OCD, with notably elevated obsessing, ordering, and checking symptoms (Jiujias et al., 2017). The high rate of comorbid psychiatric illness in the ASD population, including depression and anxiety, significantly contributes to the increased risk for Substance Use Disorders.

B.4. Role of Genetics

There are no current genetic markers or tests that confirm a diagnosis of idiopathic ASD. Genetic testing is only used to rule out syndromal forms of ASD, not to confirm an ASD diagnosis. Rather, the large number of genetic variables increases risk and vulnerability to environmental

insult. Genes account for up to about 85 percent of autism's heritability, but only about 10 percent of individuals with ASD have an identifiable genetic cause (*'Polygenic Risk Scores' for Autism, Explained*, 2023).

Common variants working together may account for almost half of autism's genetic basis, and each person has a unique set of variants that affects their autism risk. The multiple-hits theory of autism can explain why the same mutation can lead to different sets of traits in two people, as environmental insults during various times of development (in utero, early childhood, adolescence) causes changes in gene expression at the epigenetic level (*The Multiple Hits Theory of Autism, Explained*, 2019).

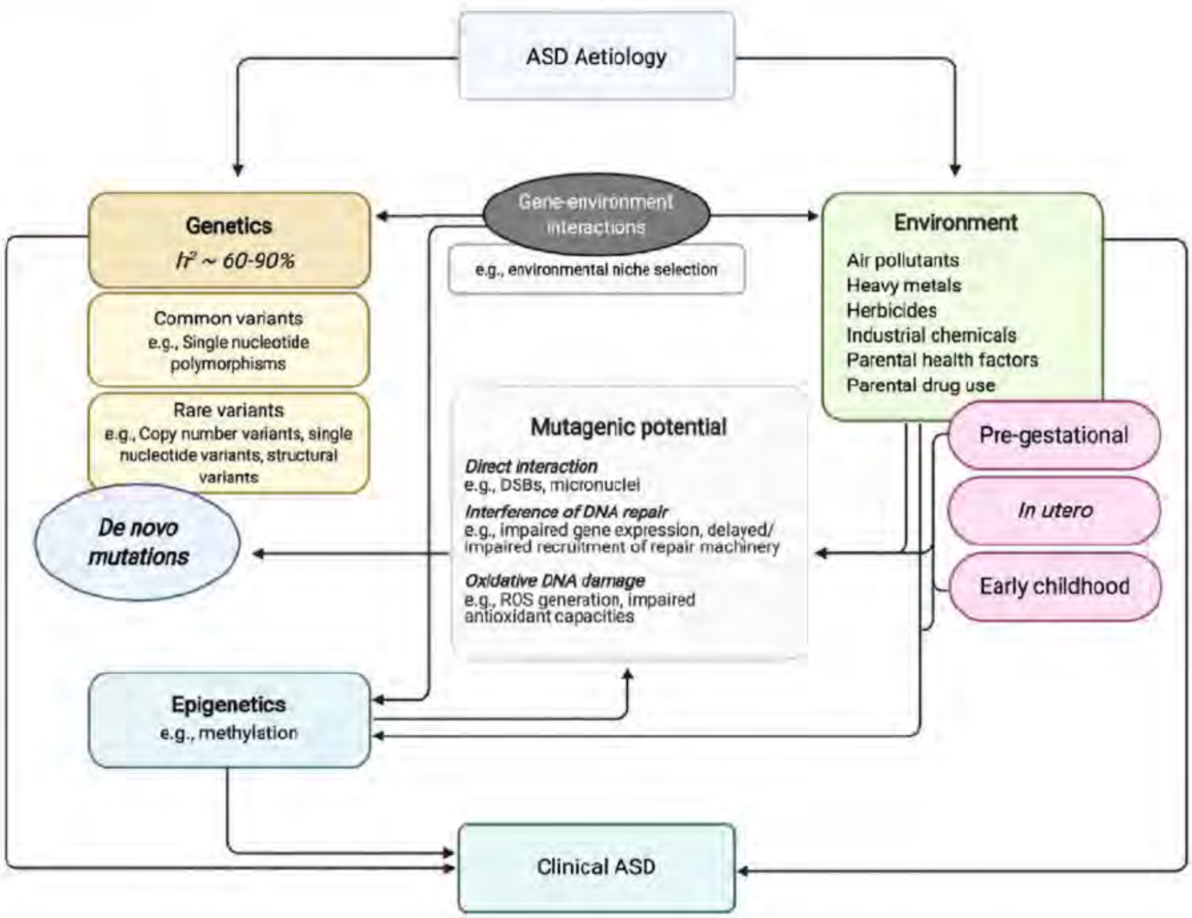


Fig. 1. Diagrammatic representation of the interplay between genetic and environmental risk factors in etiology of ASD (Pugsley et al., 2022).

Due to the heritability of ASD, siblings of individuals with ASD may be at a higher risk of developing ASD or having another neurodevelopmental or psychiatric illness (Jokiranta-Olkoniemi et al., 2016; Shephard et al., 2017). Family members of those with ASD are at a higher risk of having learning and language problems, and mood and anxiety disorders (Jokiranta-Olkoniemi et al., 2016; Sandin et al., 2014; Volkmar et al., 2014). There are also higher rates of gastrointestinal and sleep disorders in the siblings and parents of those with ASD (Aldinger, Lane, Veenstra-VanderWeele, et al., 2015).

During clinical assessment, genetic testing may be completed to rule out syndromal causes of ASD, including Fragile X Syndrome, Rett Syndrome, Tuberous Sclerosis Complex, Prader-Willi Syndrome and Angelman Syndrome. These syndromes affect 10% of individuals with a known identifiable genetic cause of ASD.

Although a large fraction of ASD's genetic roots comes in the form of single nucleotide polymorphisms (SNPs), each variant confers only a small increase in a person's chance of having ASD. Assays may be completed in research studies to scan for SNPs in those with ASD to determine which occur the most frequently. The polygenic risk score, the weighted sum of all the common variants, can then be calculated with the hope that in the future this will aid in the diagnosis of ASD. However, much more work needs to be completed before this is of use in clinical practice. Over 500 genes have been associated with ASD, with 58 common variants identified within 27 genes. Common variants explain 40% to 60% of the heritability of ASD.

B.5. Environmental Factors

Recent studies suggest that exposure to environmental factors may account for up to 40% to 50% of the variance in ASD liability. Liability is defined as all the genetic and environmental factors that contribute to the development of a multifactorial disorder, like ASD. There are

variations in the amount of evidence for each environmental factor, with some supported by association, in vitro and in vivo studies, and others with weaker evidence. Environmental factors can be divided into prenatal exposures and maternal conditions, perinatal risk factors, and parental risk factors.

Prenatal exposures and maternal conditions include, but are not limited to, maternal depression and autoimmune disease, exposure to antidepressants and anticonvulsant drugs in utero, gestational diabetes, fetal distress and cesarean delivery, viral and bacterial infections, prenatal APAP exposure, metal exposure, and decreased exposure to folic acid and other macronutrients in utero. It is difficult to separate the potential effects of SSRIs on the fetus from the mother's underlying psychiatric condition, including depression or untreated depression. Systematic reviews and metanalyses have indicated that the maternal use of SSRIs could be associated with as high as a 50% increase in ASD risk, but maternal mental health could not be removed as a confounding factor (Kobayashi et al., 2016; Andalib et al., 2017). It is clear that serotonin metabolism and altered serotonergic pathways are associated with ASD risk (Anderson et al., 2008), and animal models exposed to prenatal SSRIs have demonstrated adverse neurodevelopmental outcomes, indicating they might have a causal role. However, genetic susceptibility due to underlying maternal mental health is also supported by the continued increased risk of psychiatric disorders in children whose mothers had discontinued antidepressant use during pregnancy (Liu et al., 2017). Thus, although an increased risk of psychiatric disorders is observed in children exposed to antidepressants in utero, it is suspected this is largely due to the severity of the mother's underlying psychiatric disorder, as they are more likely to continue treatment during pregnancy.

Epidemiological studies have also shown that children exposed in utero to valproic acid, an antiepileptic and mood stabilizer, in the first trimester are at higher risk of developing ASD. These results have been further confirmed by both animal and human studies, and rodents exposed to valproic acid have symptoms that mirror those observed in their human counterparts with ASD, including social impairments, repetitive behaviors and cognitive inflexibility (Chaliha et al., 2020).

Maternal autoimmune disease or exposure to viral and bacterial infections during pregnancy are also associated with an increased risk of ASD in offspring. The elevated inflammatory markers and antibodies in pregnant women in response to an infection are noted to interfere with in utero development, increasing ASD risk. There is also an association between ASD and family history of autoimmune diseases. The maternal immune activation (MIA) hypothesis confirms the importance of the maternal immune system and the increased risk of ASD and other psychiatric/neurodevelopmental disorders in offspring (Wu et al., 2015; Comi et al., 1999; Atladóttir et al., 2009; Chen et al., 2016).

An umbrella review conducted by Kim et al. systematically evaluated relevant meta-analyses to study the strength and validity of certain environmental risk factors or biomarkers of ASD (Kim et al., 2019). The authors identified 46 eligible articles on 52 biomarkers and 67 environmental risk factors. The authors' analysis provided *convincing evidence* (Class I) in relation the association between ASD and maternal factors, including age, and features of metabolic syndrome, and the use of antidepressants like SSRIs. Additionally, the authors' analysis provided *highly suggestive evidence* (Class II) showing associations between ASD and higher paternal age, maternal exposure to autoimmune disease, and APAP exposure during pregnancy. The authors stated, however, that the results should be interpreted with caution given the statistical methods and biased tests used.

A meta-analysis of case-control studies found an increased risk of ASD of 62% in offspring with mothers with diabetes compared to those without (Wan et al., 2018). Additionally, gestational diabetes has also been associated with an increased risk of ASD, in addition to other adverse pregnancy outcomes. It is hypothesized that the oxidative stress and hormonal and metabolic abnormalities caused by gestational diabetes interferes with in utero development (Gardener et al., 2009; Xu et al., 2014). Parental age may be a confounder in the relationship of ASD risk to maternal pregestational or gestational diabetes.

Fetal distress is associated with multiple adverse outcomes, one of which may be an increased risk of developing ASD. Hypoxia, related to fetal distress, prolonged labor, cord complications, low Apgar score, maternal hypertension and cesarean delivery, are shown to increase dopaminergic activity, which could be related to dopaminergic overactivation in ASD, and increased ASD risk, although more work needs to be done examining this area (Gardener et al., 2009). Cesarean delivery has also been modestly associated with ASD in multiple studies (Curran et al., 2015).

Both increased exposure to toxic metals, and decreased exposure to those important to development, have also been suggested as playing a role in the pathogenesis of ASD. In a large mother-child cohort study, maternal erythrocyte lead concentrations in the second trimester were associated with an increase in the parent-related emotional problems subscale of the Strengths and Difficulties Questionnaire (SDQ) in both sexes, and in the total SDQ score in girls when assessed in mid-childhood (Campbell et al., 2021). Exposure to high levels of inorganic mercury has also been linked to an increased risk of ASD in offspring (Yoshimasu et al., 2014). There is also some research examining the effect of unbalanced metal levels on synapse formation, and the possible

therapeutic role of zinc supplementation (Hagmeyer et al., 2015), and the possibility it can reduce the risk of neural tube defects in offspring (Velie et al., 1999).

Exposure to organophosphates during pregnancy, including non-persistent organic pollutants (phthalates and bisphenol A), and persistent organic pollutants (DDT, PCB, PBDE) may also be linked to increased risks of offspring developing neurodevelopmental disorders, such as ASD, but more research is needed (Shelton et al., 2014).

Prenatal diet may also play a role in the etiology of ASD, and prenatal deficiencies of micronutrients have been associated with an increased risk of ASD. Folic acid is important to in utero development, and its use prenatally has protective effects, including reducing neural tube deficits. Adequate intake of folic acid and vitamin D, and folic acid supplementation, have been associated with a lower likelihood and reduced risk of ASD in offspring (Zhong et al., 2020; Liu et al., 2021).

Perinatal risk factors include, but are not limited to, low birth weight, preterm birth, and fetal growth restriction. These are the most studied ASD risk factors. Statistically significant associations have been found between ASD risk and low birth weight, neonatal anemia, umbilical cord complications, birth injury/trauma, maternal hemorrhage, caesarean section delivery, fetal distress, and blood group incompatibility (Gardener et al., 2009; Gardener et al., 2011; Wang et al., 2017). Low birth weight (< 2500 g) is an early indicator of an issue in the intrauterine environment and is a marker for high risk of multiple later neurological, psychiatric and neuropsychological problems, including cognitive difficulties, attention difficulties, social deficits, hyperactivity and learning difficulties (Hack et al., 2005). Only two epidemiological studies have found an increased risk of ASD associated with low birth weight when other risk factors are controlled for (Maimburg and Vaeth, 2006; Schendel and Bhasin, 2008). Preterm births often also

have low birth weight, and have been associated with developmental delays, intellectual impairments, and adverse health outcomes (Moster et al., 2008; Schothorst & van Engeland, 1996; Wood et al., 2000). One study demonstrated a seven-fold increase in ASD risk when the child was born before 31 weeks (Moster et al., 2008), while others have shown an increased risk at birth before 37 weeks after controlling for confounders (Durkin et al., 2008; Larsson et al., 2005). Growth restriction, a child being born small for their gestational age, is often due to factors that reduce in utero growth, including placental problems, maternal nutritional problems, and maternal infections. Three studies have found a significant association between growth restriction and ASD risk, with one study continuing to show a strong link even when preterm birth, low birth weight, and other risk factors were accounted for (Hultman et al., 2002; Larsson et al., 2005; Maimburg & Vaeth, 2006).

Parental risk factors include, but are not limited to, advanced maternal and paternal age, and maternal obesity. One of the most established environmental risk factors for ASD is parental age, and this environmental risk factor has also been linked to the development of schizophrenia, bipolar disorder and ADHD. A 10-year increase in maternal and paternal age has been associated with a 20% higher risk of ASD in children (Wu et al., 2017). Maternal age remains an independent risk factor for ASD even after adjustment for other potential confounders (Croen et al., 2007; Maimburg & Vaeth, 2006; Janecka et al., 2019; Grether et al., 2009; Durkin et al., 2008; Sandin et al., 2016; Glasson et al., 2004), and if all studies are taken into account it is possible that it increases the risk of ASD by approximately 50% when other confounders are also accounted for. Advanced paternal age has been associated with age-related methylation changes in sperm that increases ASD risk in offspring, and ASD offspring of older fathers are shown to have a reduced cortical thickness

in the right posterior ventral cingulate cortex (Atsem et al., 2016; Kojima et al., 2019). These results are supported by mouse models (Foldi et al., 2010; Sampino et al., 2014).

Meta-analyses have shown between a 28% and 36% increased risk of ASD in children with overweight and obese mothers, with a potential linear dose-response relationship between maternal BMI and risk of ASD (Wang et al., 2016). This connection still needs to be studied, as in another large study, the sibling analysis did not reveal any association between elevated BMI and ASD risk, even though there was a relationship at the population level (Gardner et al., 2015).

B.6. Major Hypotheses on Pathophysiology

The etiology of ASD is complex and involves genetic, epigenetic, environmental, metabolic, and immune-inflammatory factors (Hsiao et al., 2013; Loke et al., 2015). Metabolomic and lipidomic levels are altered in ASD (Aran et al., 2019; Brigandi et al., 2015; Likhitweerawong et al., 2021; Ming et al., 2012; Needham et al., 2020, 2021; Orozco et al., 2019; Smith et al., 2020; Tamiji & Crawford, 2011; Yui et al., 2016; Zou et al., 2021), and may be correlated with symptom severity. The polyunsaturated fatty acid (PUFA) metabolism that results in the production of eicosanoids may be overactive in ASD (Brigandi et al., 2015; Tamiji & Crawford, 2011).

This association between lipid metabolism and ASD is also supported by significant alterations of plasma omega-3 and omega-6 fatty acids, resulting in an imbalance in the ratio of omega-3/omega-6 PUFAs (Yui et al., 2016). This is important because APAP and its active metabolites, including AM404 impact lipid signaling in the CNS. Key metabolic pathways are also affected (Likhitweerawong et al., 2021; Ming et al., 2012; Needham et al., 2021; Orozco et al., 2019; Smith et al., 2020). The excitation/inhibition imbalance hypothesis of ASD may also be linked to metabolomics/lipidomics, as elevated lysophosphatidylinositol (LPI) induces cortical excitability due to its pro-excitatory and anti-inhibitory effects (Anavi-Goffer et al., 2012). Thus,

APAP and its active metabolite AM404 impact E/I imbalance due to its effects on lipid signaling pathways. Cortical excitability is also linked to other disorders characterized by inflexible thinking, like OCD (Greenberg et al., 2000).

Cortical excitability may be linked to altered cortical pathology including patchy disruption of cortical mini-columns in the frontal and temporal cortex, resulting in E/I imbalance, and manifest in increased propensity of seizures, hyperactivity, impulsivity and irritability, as described below in E/I imbalance. Individuals with intensely upregulated immune and metabolome genes in postmortem brain tissue may have a neuroinflammatory condition in which atypical microglial activation derails normal neurogenesis and synaptogenesis across various conditions, including ASD and other neurological and psychiatric conditions (Y. Chen et al., 2022). Most of the expression changes observed in brain tissue samples do not appear in gene expression patterns in blood samples from people with the same conditions.

These changes may not reflect the specific disorders, but rather a transdiagnostic endophenotype that cuts across conditions. There is significant evidence for immune dysfunction in ASD, including systemic inflammation, cytokine dysregulation and anti-brain autoantibodies (Lyall et al., 2014; Masi et al., 2017; Meltzer & Van de Water, 2016). Both prenatal and postnatal exposures to immune triggers may lead to an altered immune system and thus altered neurodevelopment (Fox et al., 2012). Individuals with ASD have impaired immune system regulation and heightened inflammatory processes demonstrated by altered cytokine and chemokine profiles (Ashwood et al., 2011) and increased microglial activation (Careaga et al., 2010; Goines & Ashwood, 2013; Hsiao, 2013; Masi et al., 2017; Onore et al., 2012; Pardo et al., 2005). Metabolomic and lipidomic levels are also altered in ASD (Aran et al., 2019; Brigandi et al., 2015; Likhitweerawong et al., 2021; Ming et al., 2012; Needham et al., 2020, 2021; Orozco et

al., 2019; Smith et al., 2020; Tamiji & Crawford, 2011; Yui et al., 2016; Zou et al., 2021), *and may be* correlated with symptom severity. The polyunsaturated fatty acid (PUFA) metabolism that results in the production of eicosanoids, inflammatory mediators that stimulate and modulate cytokine levels, may be overactive in ASD (Brigandi et al., 2015; Tamiji & Crawford, 2011). This association between lipid metabolism and ASD is also supported by significant alterations of plasma omega-3 (n-3) and omega-6 (n-6) fatty acids, resulting in an imbalance in the ratio of omega-3/omega-6 PUFAs (Yui et al., 2016).

Thus, APAP and its active metabolite NAPQI, which impacts inflammatory mediators may exert its effects via these mechanisms. Key metabolic pathways are also affected in those with ASD, including the end products of glycolysis (lactate/pyruvate), amino acid and TCA cycle metabolism and the carbon metabolism via folic acid-folate cycle (Likhitweerawong et al., 2021; Ming et al., 2012; Needham et al., 2021; Orozco et al., 2019; Smith et al., 2020). These studies also showed alterations in fatty acid oxidation and metabolites derived from gut bacteria metabolism of amino acids, carbohydrates, and bile acids. Elevated levels of pro-inflammatory cytokines, anti-inflammatory cytokines, and chemokines are linked to more pronounced ASD stereotypical behaviors (Ashwood et al., 2011a, 2011b). Additionally, the use of immunomodulatory treatments in ASD have experimentally shown to reduce RRBs, including cognitive and behavioral rigidity as measured by the Montefiore Einstein Rigidity Scale – Revised (MERS-R) (Hollander, Uzunova, et al., 2020).

One of the commonly proposed mechanisms of the deficits in ASD is an increased excitatory-inhibitory (E/I) ratio and imbalance between excitation and inhibition. E/I imbalance is thought to be due to abnormal GABAergic and glutamatergic neurotransmission in key brain regions, including the parietal-occipital and frontal cortical regions (Uzunova et al., 2016).

Abnormalities in E/I balance in these regions can result in seizures, in addition to behavioral changes and social dysfunction, including irritability, repetitive and disruptive behaviors, and social avoidance and withdrawal. APAP and its active metabolite AM404 exert effects on E/I imbalance and glutamate/GABA activity, and may cause ASD via this mechanism.

A common hypothesis for the etiology of ASD is aberrant excitatory-inhibitory (E/I) neurocircuitry. This imbalance is shaped by a combination of local hyperconnectivity and long-range hypoconnectivity primarily through GABA and glutaminergic pathways. These abnormalities have been localized to regions such as the hippocampus, cerebellum, amygdala, and neocortex (Nezgovorova et al., 2021; Uzunova et al., 2016). E/I imbalance may be due to increase in glutamatergic or decrease in GABAergic signaling. Cortical mini-columns involved in E/I imbalance are pathologically altered in ASD and consist of neuronal aggregations and their afferent, efferent and inter-neuronal connections, mediating the interactions of neuronal microcircuits (Casanova, 2007; Opris & Casanova, 2014; Uzunova et al., 2016). Cortical mini-columns (also known as microcolumns) are vertical columns through the cortical layers of the brain that are essential elements for cortical information processing. These structural modular arrangements of connected networks of neurons are fundamental computational units of the cerebral cortex.

ASD is proposed to be a "minicolumnopathy" with increased numbers of mini-columns and decreased mini-column width which leads to the sheath around the mini-columns being decreased due to dense packing and allowing for less inhibitory control. These aberrant neuronal connections in subependymal and subcortical regions may cause seizures, developmental delay, dyslexia, OCD and ASD (Casanova, 2007, 2008; Casanova et al., 2003; Uzunova et al., 2016). Key metabolic pathways were affected in ASD studies, such as end products of glycolysis

(lactate/pyruvate), amino acid and TCA cycle metabolism (Likhitweerawong et al., 2021; Ming et al., 2012; Needham et al., 2021; Orozco et al., 2019; Smith et al., 2020) and one carbon metabolism (Homocysteine metabolism/methionine cycling) via folic acid-folate cycle (Orozco et al., 2019). Significant metabolites derived from gut bacteria metabolism of amino acids, carbohydrates, and bile acids were altered in children with ASD (Ming et al., 2012; Orozco et al., 2019) and fatty acid oxidation (Needham et al., 2021). As mentioned above, association between lipid metabolism and ASD is supported by significant alterations of omega-3 (n-3) and omega-6 (n-6) fatty acids seen in the plasma of ASD patient including the ratio of omega-3/omega-6 PUFAs. Differentially expressed lipids in ASD include phospholipids, cholesterol esters and glycerolipids, in general shorter saturated fatty acid chain (14-18) were less abundant in ASD and elevations in PUFA side chains were seen in DAGs, and enrichment of 18:2 linolenic acid in most lipid classes (Needham et al., 2021). PUFA metabolism, resulting in production of eicosanoids, may be overactive in ASD.

Individuals with ASD have marked immune dysfunction and heightened inflammatory processes demonstrated by altered cytokine and chemokine profiles (Ashwood et al., 2011) and increased microglial activation (Goines & Ashwood, 2013; Hsiao, 2013; Masi et al., 2015; Onore et al., 2012; Vargas et al., 2005). Individuals with ASD have elevated pro-inflammatory cytokines (IL-1β, IL-6, IL-8, IL-12p40) (Ashwood et al., 2011) and IFN-γ and decreased anti-inflammatory cytokines (IL-10 and TGFβ). Increases in the pro-inflammatory cytokines are associated with more regressive autism and more pronounced stereotypical behaviors. Further, in children with ASD and asthma IL-17 is elevated following T-cell stimulation (Akintunde et al., 2015). These changes in cytokines in ASD may be developmentally regulated as they differ when measured during the neonatal period compared to later developmental periods (Abdallah et al., 2012; Abdallah, Larsen, Grove, Bonefeld-Jørgensen et al., 2013; Abdallah, Larsen, Grove, Nørgaard-Pedersen, et al., 2013;

M. L. Estes & McAllister, 2015). Seizures are frequent among children with ASD and may also have an association with immune dysfunction, inflammation and altered cytokines. Polyunsaturated fatty acid (PUFA) metabolism, resulting in production of eicosanoids, inflammatory mediators which can be stimulated by, and in turn modulate, cytokine levels, may be overactive in autism (Brigandi et al., 2015; Tamiji & Crawford, 2011; Yui et al., 2016). In addition, an imbalance in the ratio of omega-3/omega-6 PUFAs is noted in ASD.

Data from the Autism Brain Imaging Data Exchange has also shown differential associations between BMI and brain structural changes in children with ASD compared to typically developing children. BMI is negatively associated with left caudate volume (a part of the human brain involved in movement and cognitive function) and positively associated with bilateral ventral diencephalon volumes (a part of the human brain that coordinates signals to the endocrine system and cerebral cortex). These changes in subcortical volumes were also significantly associated with ASD symptom severity, as measured by the Vineland (Hwang & Hong, 2022). Proposed mechanisms for the links between obesity, ASD and symptom severity include hyperactivation of the PI3K/AKT/mTOR pathway (involved in regulating the cell cycle), insulin signaling and endocrine markers, including leptin. The PI3K/AKT/mTOR pathway affects synaptic plasticity, and an increase in mTOR is shown to reduce synaptic plasticity. Insulin is able to cross the blood-brain barrier, and regulates synaptic activity in the cerebellum, prefrontal cortex and hippocampus, in addition to other areas. Insulin signaling in the brain is predicted to activate the PI3K/AKT/mTOR pathway. It is possible that for mothers with diabetes insulin signaling hyperactivates the fetal PI3K/AKT/mTOR pathway causing structural and signaling changes very early in development that lead to ASD (Stern, 2011).

The endocannabinoid (eCB) system has an important role in neurodevelopment (Basavarajappa et al., 2009) and is transiently activated during stressful conditions (Steiner & Wotjak, 2008). It exerts its effects through G-protein coupled (GPCR) CB_1 and CB_2 cannabinoid receptors, transient receptor potential (TRP) channels (Iannotti et al., 2014) that may modulate calcium flux (Ryan et al., 2009), the orphan G-protein-coupled receptor GPR55, the 5-HT1A receptor, the α3 and α1 glycine receptors (Devinsky et al., 2014; Li, Jones, & Persaud, 2011), and nuclear PPARs (Battista et al., 2012). CB_1 receptors are among the most widely expressed GPCRs in the brain and may occur in peripheral nerves and non-neuronal tissues. CB_2 receptors are expressed predominantly in cells of the immune system (Siniscalco et al., 2013), and may be expressed at sites of tissue damage, including within the central nervous system (CNS). eCBs are synthesized on demand and the eCB system may be modulated by ligand binding to the CB_1 and CB_2 receptors, inhibition of cellular eCB uptake or by modulating the intracellular metabolism of eCB by specific enzymes (Battista et al., 2012).

Numerous studies implicate the eCB system in ASD (Chakrabarti et al., 2015). Disruption of this system may impair social communication, social play and reciprocity (Kerr et al., 2013). Polymorphisms in the CB_1 receptor gene may adversely affect social reward processing in ASD (Chakrabarti & Baron-Cohen, 2011). The BTBR autism mouse has upregulated CB2A gene expression in the cerebellum, and treatment with an eCB reduces locomotor activity, suggesting an impact on irritability and repetitive behaviors (Onaivi et al., 2011). Children with ASD have increased CB_2 mRNA and protein levels in peripheral blood compared to healthy subjects (Siniscalco et al., 2013). Intriguingly, eCB-mediated signaling at inhibitory synapses is dysregulated in mouse models of autism-associated Neuroligin 3 mutations (Földy, Malenka, & Sudhof, 2013). In another mouse model of autism, the Fragile X knockout mouse, there is an

absence of an eCB-mediated type of synaptic plasticity (long term depression) in the ventral striatum and prefrontal cortex. Pharmacological enhancement of the eCB signaling normalizes the synaptic plasticity deficit and corrects the behavioral abnormalities, suggesting that the eCB signalosome is a molecular substrate in Fragile X syndrome (Jung et al., 2012).

eCBs also have potent anti-inflammatory and immunosuppressive properties (Devinsky et al., 2014; Jean-Gilles, Gran, & Constantinescu, 2010; Klein & Cabral, 2006; Siniscalco et al., 2014). eCBs have been identified in immune cells, such as monocytes, macrophages, basophils, lymphocytes, and dendritic cells (Cabral, Rogers, & Lichtman, 2015). Reciprocal regulation has been described between the eCBs and the immune system mediated by cytokines (Jean-Gilles, Gran, & Constantinescu, 2010). Children with ASD have immune system dysfunction including marked microglial activation (Takano, 2015), altered cytokine profiles (Masi et al., 2015) with an elevation of pro-inflammatory cytokines in the postmortem brain and peripheral blood (McDougle et al., 2015), the presence of autoantibodies directed to brain and other antigens, and an association with MHC complex haplotypes (Gesundheit et al., 2013). From these immune system changes, peripheral blood cytokines are readily measurable and quantifiable biomarkers (Rose & Ashwood, 2014). Elevated levels of the enzyme Nagalase, which is responsible for proper macrophage function (via Gc Protein-Derived Macrophage Activating Factor, GcMAF) (Bradstreet et al., 2012), and significantly upregulated CB_2 receptor mRNA in peripheral blood mononuclear cells (PBMC) have been reported in children with ASD (Siniscalco et al., 2013). Decreased serum levels of N-arachidonoylethanolamine (AEA or anandamide) and N-palmitoylethanolamine (PEA), and N-oleoylethanolamine (OEA) have been found in two case-control studies in ASD children compared with matched healthy children (Karhson et al., 2018; Aran et al., 2019). One case control study also found decreased plasma AEA, PEA and OEA levels as well as a decrease in plasma 2-

arachidonoylglycerol (2-AG) in ASD children (Zou et al., 2021). These alterations indicate that the eCB system is involved in ASD pathogenesis.

Treatment with GcMAF ameliorates the autistic symptoms in some children (Siniscalco et al., 2014). This may be due to effects on the gene expression of the eCB system and CB2R protein, and down-regulation of the over-activation of blood monocyte-derived macrophages. The multiple links between the eCB system and ASD suggest mechanisms and targets for potential treatments.

ASD studies show key metabolic pathways are affected, such as end products of glycolysis (lactate/pyruvate), amino acid and TCA cycle metabolism (Smith et al., 2020; Ming et al., 2012; Orozco et al., 2019; Needham et al., 2021; Likhitweerawong et al., 2021) and one carbon metabolism (Homocysteine metabolism/methionine cycling) via folic acid-folate cycle (Orozco et al., 2019). Significant metabolites derived from gut bacteria metabolism of amino acids, carbohydrates, and bile acids were altered in children with ASD (Ming et al., 2012; Orozco et al., 2019).

Oxidative stress, caused by an imbalance of prooxidative and antioxidative substances that results in the increased production of cell-damaging free radicals, is also important in the pathophysiology of ASD and related disorders. Fetuses are highly susceptible to the reactive oxygen species (ROS) produced by oxidative stress, and multiple prenatal risk factors for ASD development in offspring, including exposure to APAP, low birth weight, and preeclampsia, are also linked to oxidative stress. This suggests that prenatal oxidative stress directly impacts fetal neurodevelopment, and/or indirectly acts through other mechanisms, such as inflammation or placenta dysfunction (Carey et al., 2022).

Biomarkers of oxidative stress include, but are not limited to, 8-oxo-deoxy-guanine (8-OHdG), glutathione (GSH), glutathione disulfide (GSSG), GSH:GSSG ratio, 3-nitrotyrosine, and

prostaglandin F2alpha (PGF2a), free-8-isoprostane-prostaglandin-F2alpha. In a study examining oxidative stress biomarkers in pregnant women who already had a child with ASD, and in their offspring at 36 months (EARLI study), modest increases were observed in ASD-related traits with increasing GSH:GSSG ratio, a measure of antioxidant balance, for those in the middle percentiles of Social Responsiveness Scale (SRS, measure of social communication deficits) (Carey et al., 2022). Increasing levels of the GSH:GSSG ratio indicate less oxidative stress and increased antioxidant balance. There was also evidence supporting an inverse association between risk of ASD or non-typical development, with greater antioxidant balance (higher GSH:GSS ratio), which suggests the association between ASD symptoms and oxidative stress exists across a spectrum, and that increased oxidative balance prenatally, results in reductions in ASD or non-typically developing symptoms in offspring.

The presence of oxidative stress biomarkers during pregnancy and increased ASD risk in offspring is also supported by a study examining the associations between multiple urinary biomarkers of oxidative stress in the 3rd trimester of pregnancy and behavioral development in children (Infant Development and the Environmental Study, TIDES; Rommel et al., 2020). These researchers found a 2.58% increase in child SRS T-scores at age 4-5 associated with an IQR increase in levels of free 8-isoprostaneprostaglandin-F2alpha in their mother's urine samples in the third trimester. This association was modified by level of maternal education. Scores on the BASC-2 externalizing problems subscale at age 4 years were also positively associated with third trimester urinary concentrations of PGF2alpha, and were also modulated by level of maternal education. The BASC Behavioral Symptoms Index in children at age 4, was also positively associated with third trimester urinary concentrations of 8-iso-PGF2alpha and PGF2alpha. These results suggest that oxidative stress during the third trimester is associated with social impairments

and behavioral problems in children, that correlate with symptoms of ASD, ADHD and related disorders. Of note, while the EARLI study is one of pregnant mothers who already have a child with ASD, the TIDES study was completed in the general population. As pregnant women are already more susceptible to oxidative stress, and are found to have lower levels of glutathione (Balasubramanian & Birundha, 2019), the primary intracellular ROS antioxidant and detoxification mechanism in the body, any additional environmental or genetic insult may exacerbate this resulting in aberrant fetal neurodevelopment.

Biomarkers of oxidative stress have also been found in children with ASD, suggesting that in utero exposure to oxidative stress causes pathophysiological changes that persist across the child's lifespan. A study of plasma metabolites in the methionine cycle and transsulfuration pathway in 20 autistic children and 33 neurotypical controls, showed that children with autism had significantly lower baseline plasma concentrations of methionine, SAM, homocysteine, cysteine and total glutathione, and higher concentrations of SAH, adenosine and the oxidized disulfide form of glutathione (GSSG) (James et al. 2004). These results were replicated in a larger study of 80 children with ASD compared to those who are typically developing (n = 73), children with ASD had significantly decreased levels of methionine, S-adenosylmethionine (SAM), SAM/SAH ratio, cysteine, total glutathione, free reduced glutathione, and GSH:GSSG ratio, and had increased levels of S-adenosylhomocysteine (SAH), cystathionine and the oxidized disulfide form of glutathione (GSSG) (James et al., 2006). These key metabolic abnormalities indicate significant impairments of antioxidant capacity, redox homeostasis, and methylation capacity, and that these individuals with ASD were currently experiencing oxidative stress.

The autism Integrated Metabolic and Genomic Endeavor (IMAGE) study is a case-control study with over 68 case children, 40 unaffected siblings, and 54 age-matched unaffected control

children, examining the role of metabolic and genomic factors in the pathophysiology of ASD and symptom severity (Melnyk et al., 2012). Similar to the results for the previous two studies discussed, children with autism were found to have significantly decreased methionine and SAM, and significantly increased SAH and adenosine, compared to their paired unaffected siblings. Total cysteine levels and the GSH:GSSH ratio were also decreased in children with autism compared to their unaffected siblings indicating increased oxidative stress. On the majority of the above markers, unaffected siblings were not different from unrelated controls. The authors discuss the important role of gene-environmental model and how the epigenetic alterations and oxidative DNA damage observed in the children with autism in this study, is consistent with the Latent Early-life Associated Regulation (LEARn) model. The LEARn model proposes that environmental and genetic risk factors operate through oxidative stress and alterations in DNA methylation in susceptible genes which ultimately results in altered gene expression. Epigenetic and redox alterations are seen as dynamic adaptive responses to environmental stressors. Studies of frozen brain tissue from individuals with autism and unaffected controls confirms the above results (Rose et al., 2012). Biomarkers of oxidative stress, including significantly decreased GSH and GSH/GSSG ratio and significantly increased 3-nitrotyrosine (3-NT) and8-oxo-deoxyguanosine (8-oxo-DG) levels, were found in the cerebellum and temporal cortex of brains of individuals with autism compared to controls. These results continue to support the role of increased oxidative stress in ASD. Biomarkers of oxidative stress are noted to continue into adulthood for those with ASD and are correlated with autism symptom severity as measured by the Autism Quotient score (Thorsen et al., 2022). In a study of adults with ASD compared to age- and sex-matched controls, individuals with ASD had higher levels of the antioxidant superoxide dismutase 1 (SOD1) which was correlated with higher scores on the Autism Quotient.

B.7. Pharmacological and Psychological Treatments

To date, no medication has been approved to treat the “core” symptoms of ASD. Instead, pharmacologic treatment aims to manage the comorbid behavioral symptomatology that is often associated with ASD including irritability, hyperactivity, aggression, impulsivity, anxiety, and affective symptoms. Medications are being developed for individuals with ASD that specifically target the mechanisms thought to be involved in the pathophysiology of the core symptom domain of social communication and repetitive behaviors (i.e., oxytocin and vasopressin-related treatments) (Hollander & Uzunova, 2017). More importantly, however, various psychological treatment options are available for individuals with ASD. The most studied and empirically supported behavioral treatment is Applied Behavioral Analysis (ABA), which utilizes discrete trials to teach simple skills with a gradual progression to more complex skills as the individual’s behavior improves (Roane et al., 2016). Speech therapy is particularly important for those with ASD who are nonverbal, as it can be used to teach them alternate modes of communication. In addition, other forms of therapy such as Cognitive Behavior Therapy (CBT) and Dialectical Behavioral Therapy (DBT) may be recommended. Because those with ASD are less likely to respond to CBT, alternative ways of presenting the intervention have been created to better accommodate the symptom presentation of those with ASD. DBT is a comprehensive, evidence-based form of psychotherapy that evolved from CBT as a way to help individuals with severe emotion dysregulation. Mindfulness therapies have also been used with individuals with ASD across the lifespan, with modifications made to address common inattention, self-awareness difficulties and abstraction limitations present in the population (Beck et al., 2020). Occupational therapy is a commonly prescribed treatment for children with ASD. Residential treatment and

group homes may be considered when the individual presents with severe behavioral symptoms that cannot be addressed in outpatient settings.

C. Attention-Deficit Hyperactivity Disorder

ADHD is a chronic neurodevelopmental disorder characterized by persistent patterns of inattention and/or hyperactivity and impulsivity that are disruptive and result in functional impairment in multiple settings. Behaviors must be present for at least 6 months, and must have started before age 12. The symptoms must be present across more than one setting (i.e., home, work, or school), and these symptoms must result in academic, social, or occupational impairment. ADHD symptoms may be more difficult to detect in females, due to more predominant inattention symptoms in early childhood that do not cause as significant of functional impairment until early adolescence. When diagnosing individuals aged 17 and older, fewer symptoms are required.

ADHD is the most frequently occurring and researched psychiatric disorder of childhood and accounts for the majority of referrals to child and adolescent psychiatry services. In the United States, 6 million (9.8%) children aged 3 to 17 years are estimated to live with ADHD, according to a national survey of parents. Boys (13%) are more likely to be diagnosed with ADHD than girls (6%). Approximately 6 in 10 children with ADHD had at least one other mental, emotional or behavioral disorder, with half also diagnosed with a behavior or conduct problem, and about 3 in 10 diagnosed with comorbid anxiety. Other common comorbid disorders are ASD, depression, and Tourette syndrome. ADHD persists into adulthood in many individuals, and the burden of the disease in adults has been well described. Meta-analyses estimate the prevalence of ADHD in adults aged 19 to 45 years to be between 2% and 5% (Simon et al., 2009), with approximately 15% of patients persisting with full diagnostic criteria in adulthood and 40% to 60% classified as partial remitters (Posner et al., 2020).

Point prevalence rates for ADHD are similar across the globe, including North America, Europe, Oceania, South America, Asia, Africa, and the Middle East. Community samples of children and adolescents from 35 countries in six continents show a prevalence rate of ADHD ranging from 5% to 29% (Polanczyk et al., 2014). Over the past two decades, rates of children diagnosed with ADHD have increased significantly from 7% to 10.2% (Ji, 2018). As with ASD, there have been statistically significant increases in the proportion of children ages 5 to 17 years reported to have been diagnosed with ASD and/or ADHD between 1997 and 2017 (U.S. EPA, "Health – Neurodevelopmental Disorders," Overviews and Factsheets, May 29, 2015). Prevalence rates do not differ between North America and Europe, which supports the view that ADHD is not a construct of North American culture. Within North America, the National Survey of Children's Health (NSCH) regularly completes population-based surveys examining prevalence rates of multiple neurodevelopmental, mental health, and medical disorders in addition to other aspects of children's health. Through this study, the percentage of children with a lifetime diagnosis of ADHD increased from 7.8% to 9.5% from 2003 to 2007, a 21.8% increase. From 2003 to 2011, NSCH determined that the number of children aged 3 to 17 ever diagnosed with ADHD in the US steadily increased from 4.4 million to 6.4 million. Prevalence estimates from 2016 to 2019 show that 6 million children in the US aged 3 to 17 were ever diagnosed with ADHD.

Additional studies of ADHD medication usage have demonstrated that 66.3% of children and adolescents with a current ADHD diagnosis were taking medication for the disorder, which represented 4.8% of all children aged 4 to 17 years. In California, an analysis of medical records from 2001 to 2010 showed a relative increase of 24% in the incidence of physician-diagnosed ADHD in children aged 5 to 11 (Getahun et al., 2013). These numbers are consistent with other studies that use administrative data to calculate prevalence rates in the USA, UK, and Canada from

1990s through 2000s (Bitsko et al., 2022; Cree et al., 2023; Danielson et al., 2018; G. Polanczyk et al., 2007; G. V. Polanczyk et al., 2014; Posner et al., 2020; Visser et al., 2016).

C.1. DSM-IV and DSM-5 Definitions and Diagnostic Criteria

The course of ADHD varies between individuals, and four developmental trajectories have been identified, including (1) early onset (preschool, ages 3 to 5), (2) middle childhood onset with a persistent course (ages 6 to 14), (3) middle childhood onset with adolescent offset (6 to 14 years), and (4) adolescent or adult onset (16 years and older). Within the DSM-5, the newer concept of partial remission was introduced, which acknowledges that some children diagnosed with ADHD may not have symptoms that functionally impact their activities of daily living beyond age 18. In the Longitudinal Assessment of Manic Systems (LAMS) study, 431 children with childhood-onset ADHD were monitored for eight years, with about 30% demonstrated stable full remission at the end of their participation and more than half continuing to meet ADHD criteria (Van Meter et al., 2023).

Through periodic review and reformulation, the specific criteria for children and adults to receive an ADHD diagnosis has changed to fit with growing knowledge and larger working hypotheses about the structure and nature of the disorder. In DSM-5, the changes made to the diagnostic criteria included modifying the required age of symptom onset from before age 7 (DSM-IV criteria) to before age 12, so that diagnoses could be made more effectively for adults; and changing the term "subtype" to "presentation" in an effort to recognize that symptom profiles can change across the lifespan. The complete DSM-5 and DSM-IV diagnostic criteria for ADHD are excerpted in **Appendix 2.**

These diagnostic changes have also been made in the ICD-11, where ADHD has been moved from the disruptive behavior domain to the neurodevelopmental disorder domain,

highlighting it as a disorder of the nervous system caused by both genetic and environmental effects prenatally. Additional changes to the ICD in its latest version included changing the diagnostic label from "hyperkinetic disorder" to "ADHD," including both the hyperactive-impulsive and inattention presentations, making the diagnosis uniform across international bodies.

As discussed above, ADHD is a chronic neurodevelopmental disorder that typically has onset in early childhood, although onset later in life is possible as individuals are confronted with more cognitive demands and situations that tax executive functioning resources. Hyperactivity symptoms usually begin at age 3 or 4, with combined symptoms of inattention and hyperactivity usually presenting from ages 5 to 8. Symptoms are progressive and constant.

Under the DSM-IV-TR, ADHD was classified as a disruptive behavior disorder (Doernberg & Hollander, 2016). With the introduction of the DSM-5, however, ADHD was classified as a neurodevelopmental disorder. The 18-symptom diagnosis criteria for ADHD in the DSM-IV remained nearly the same in the DSM-5 (Doernberg & Hollander, 2016). Significantly, the DSM-5 chapter for Neurodevelopmental Disorders allows for comorbid diagnosis of both ASD and ADHD, which was not previously allowed in the DSM-IV.

ADHD may also be referred to as Hyperkinetic Disorder (HKD) in some countries, and the terms may be used interchangeably. Both ADHD and HKD refer to a combination of inattention, hyperactive, and impulsive behavior in children. HKD is the term listed in the ICD-10. The complete list of elements of the ICD-10 are excerpted in **Appendix 2.**

Differences in diagnostic criteria between the DSM-5 and ICD-10 can be seen in the table below. The overall consensus diagnostically is that HKD is a subset of ADHD, which identifies a refined phenotype (Swanson et al., 1998).

Table. Comparison of DSM-5 and ICD-10 diagnostic criteria for ADHD

	DSM-5	ICD-10
Name	ADHD	Hyperkinetic disorder
Onset	Some symptoms before age 12	Some symptoms before age 6
Symptom criteria for children	ADHD combined: 6 of 9 symptoms of inattention and 6 of 9 symptoms of hyperactivity/impulsivity ADHD predominantly inattentive: 6 of 9 symptoms of inattention ADHD predominantly hyperactive/impulsive: 6 of 9 symptoms of hyperactivity/impulsivity	Must have a combination of impaired attention AND hyperactivity The only subtype is hyperkinetic conduct disorder for those who meet criteria for both disorders
Symptom criteria for persons aged >_ 17	ADHD combined: 5 of 9 symptoms of inattention and 5 of 9 symptoms of hyperactivity/impulsivity ADHD predominantly inattentive: 5 of 9 symptoms of inattention ADHD predominantly hyperactive/impulsive: 5 of 9 symptoms of hyperactivity/impulsivity	Must have a combination of impaired attention and hyperactivity
Settings	Several symptoms present in ≥ 2 settings	Full syndrome in ≥ 2 settings and observed by clinician
Duration	≥ 6 months	≥ 6 months
Impairment	Interference with social, academic, or occupational functioning; includes severity specifiers: mild, moderate, severe	Clinically significant distress or impairment in social, academic, or occupational functioning.

Table taken from "Are We Overdiagnosing and Overtreating ADHD?" Psychiatric Times, May 31, 2019.
Rahil R. Jummani, MD., Emily Hirsch, Glenn S. Hirsch, MD.

C.2. Sex Differences in ADHD Diagnoses

Overall, ADHD is more prevalent in males, at ratios of approximately 3:1, although some studies have reported smaller male to female ratios (2.28:1) due to possible underdiagnosing of ADHD cases in females (Ramtekkar et al., 2010). This disorder typically emerges during childhood or early adolescence (Faraone et al., 2021). A comprehensive meta-analysis examining parent ratings of symptoms in 29 studies with over 42,000 participants, and teacher ratings in 24 studies with over 56,000 participants, revealed an approximate two-to-one male-to-female ratio among youth (Willcutt, 2012).

However, research has increasingly shown that ADHD is not predominantly a disorder affecting males, although boys are more commonly diagnosed with ADHD compared to girls, with

ratios ranging from 2:1 to 9:1 depending on the subtype and setting (Rucklidge, 2010). It is worth noting that in clinical settings, boys are more likely to be referred for evaluation (Rucklidge, 2010). However, studies conducted with adult populations have revealed a higher prevalence of ADHD in women than in men in certain psychiatric outpatient services. Estimates suggest that globally, approximately 32 million females have ADHD, highlighting the significance of addressing ADHD in females as a major public health concern (Rucklidge, 2010).

It is important to recognize that there is still a tendency for referral bias, leading to under-identification of ADHD in females, especially among younger individuals (Rucklidge, 2010). Moreover, recent research has questioned previous assumptions that females with ADHD are less affected by the disorder compared to males (Rucklidge, 2010). Current studies have provided compelling evidence that females with ADHD face significant challenges across multiple aspects of functioning, including academics, cognition, psychosocial well-being, and mental health. These findings demonstrate that females with ADHD experience similar levels of difficulties as males with ADHD, highlighting the substantial impact of the condition on their overall functioning (Rucklidge, 2010).

Numerous studies have consistently shown that girls diagnosed with ADHD are more commonly associated with the predominantly inattentive type, in contrast to boys (Biederman et al., 2005; Weiss, et al. 2003). Additionally, parents and teachers tend to perceive boys with ADHD as more hyperactive, while girls are often seen as more inattentive (Papageorgiou, et al., 2008).

Boys diagnosed with ADHD tend to exhibit higher rates of comorbid externalizing problems, while girls with ADHD, particularly during adolescence, tend to have higher rates of comorbid internalizing problems such as depression and anxiety (Gerschon, 2002). However, it is

important to note that studies conducted on non-referred samples do not find gender differences in the rates of coexisting psychiatric disorders among children with ADHD.

Mahendiran et al. investigated potential sex differences with age in social adaptive function across ASD and ADHD (Mahendiran et al., 2019). The objective of the study was to investigate potential variations in social adaptive functioning across different ages and between males and females with ASD, ADHD, and typically developing individuals. In the ADHD group, significant findings were identified. Additionally, sex differences and interactions between age and diagnosis were observed in the social and leisure domains between ASD and ADHD. Females with ADHD consistently exhibited higher scores in social skills compared to males across all age groups. Similar patterns of sex differences in the social domains were observed between the ADHD group and the typically developing controls. Notably, differences in social and communication skills were observed between ASD, ADHD, and typically developing individuals.

To maximize the efficacy of psychosocial and medical treatments for individuals with ADHD, it is crucial to consider the varying impact of the disorder on different genders across various domains of functioning. Understanding these gender-specific effects is vital in tailoring interventions to address the unique needs of both males and females affected by ADHD.

C.3. Associated Features and Co-Morbid Psychiatric and Medical Disorders

ADHD often co-occurs with other psychiatric disorders, including depression, bipolar disorder, ASD, anxiety disorders, oppositional defiant disorder, conduct disorder, eating disorder, and substance use disorders. The presence of these co-morbid disorders does not rule out a diagnosis of ADHD. Approximately 6 in 10 children (64%) with ADHD are diagnosed with at least one other mental, emotional, or behavioral disorder. Individuals with ADHD and comorbid depression are shown to have greater functional impairment, including longer and more severe

depressive episodes and higher rates of suicidality, when compared to those with only ADHD, or only depression (Biederman et al., 2008; Chronis-Tuscano et al., 2010).

Individuals with ADHD are also at a higher risk for certain medical disorders, including obesity, asthma, diabetes mellitus, and somatic disorders. In particular, researchers found that individuals with ADHD had a threefold greater risk of obesity compared to their family members without ADHD (Chen et al., 2018). Studies show that individuals with asthma or who were born to asthmatic mothers were between 40% to 45% more likely to have ADHD (Cortese et al., 2018; Liu et al., 2019). ADHD was 40% more likely to be diagnosed among children with type 1 diabetes (Kapellen et al., 2016), and about three times more likely to develop type 2 diabetes (Chen et al., 2018).

Individuals with ADHD have a greater sleep onset latency and lower sleep efficiency (Lugo et al., 2020). Individuals with ADHD also present with greater rates of autoimmune and inflammation disorders, such as psoriasis (Hegvik et al., 2018). There is also over twice the prevalence of ulcerative colitis, autoimmune thyroid disease, and ankylosing spondylitis in those with ADHD (Chen et al., 2017). Children with an autoimmune disease were 24% more likely to develop ADHD, and those born to mothers with autoimmune disease had a 12% greater chance of developing ADHD (Nielsen et al., 2017). Celiac patients are also shown to have a 29% increased risk of ADHD (Lebwohl et al., 2020). Individuals with epilepsy have between a 2- and 4-fold increased risk of ADHD (Bertelsen et al., 2016; Chou et al., 2013; Brikell et al., 2018).

C.4. Imaging and Anatomical Differences

Meta-analyses of task-based functional MRI (fMRI) studies have identified abnormalities in the function of many neural networks, including those related to attention and executive function. Regions of inhibitory control are frequently under-activated in those with ADHD

compared to neurotypical controls, particularly the right inferior frontal cortex, basal ganglia, and supplementary motor area (Hart et al., 2013; Lukito et al., 2020, Norman et al., 2016).

Structural MRIs (sMRI) have also been used to examine brain structure, with meta-analyses confirming alterations in the limbic system and basal ganglia. Familial risk of ADHD has also been linked to reductions in total grey matter and abnormal basal ganglia volumes. Grey and white matter abnormalities are shown to persist into adulthood for some individuals with ADHD.

Individuals with ADHD also appear to have smaller hippocampus volume compared to those with OCD; and smaller intracranial volume compared to individuals with ASD or OCD (Boedhoe et al., 2020). A study of over 4,000 participants using sMRI showed children with ADHD had slightly reduced total cortical surface area, smaller frontal, cingulate, and temporal regions, and reduced volumes in the basal ganglia, amygdala, hippocampus and intracranially (Hoogman et al., 2017; Hoogman et al., 2019). Examination of the brains of those with ADHD using diffusion MRIs has shown widespread alterations in white matter microstructure, particularly in the right anterior corona radiata, right forceps minor, left cerebellum, and bilateral internal capsule. The most consistent white matter differences between those with and without ADHD are in the splenium of the corpus collosum, and can extend to the right cingulum, right sagittal stratum and left tapetum. This suggests there are problems with the posterior parieto-temporal attention regions and long-range fronto-posterior association tracts that connect the two hemispheres. Both of these regions are involved in attention and perception (Chen et al., 2016).

There is also evidence for abnormal interactions in large-scale brain networks, with maturational delay during cortical development in utero. Although research into this area is still in the early stages, there is evidence that children with ADHD present with hyperconnectivity between the dorsal attention network (DAN) and regions of the default mode network (DMN).

There is also evidence of hyperconnectivity between the DMN and regions involved in the somatosensory, visual and auditory cortices (Lin et al., 2021). While this study indicated the presence of hyperconnectivity, others have suggested there is decreased functioning connectivity in some regions of the DMN and DAN, and more research is needed to better understand how these networks are impacted in ADHD. Electrophysiological studies have shown some evidence that children with ADHD have small-to-moderate reductions in mismatch negativity amplitude compared to neurotypical controls, which could indicate issues with auditory sensory memory and involuntary attention switching (Cheng et al., 2016).

C.5. Neurobiological Markers

There is no known diagnostic neurobiological marker for ADHD. Animal models have been helpful in understanding the role of dopaminergic, noradrenergic. and serotonergic neurotransmission in the presentation of inattention, motor overactivity, and impulsivity symptoms. Examination of cerebral networks responsible for motor response inhibition, one of the most comprised cognitive functions in ADHD, have not found any way to systematically use activity in these regions to identify those with ADHD (Massat et al., 2018). Response inhibition is the ability to inhibit the initiation or the execution of inappropriate but prepotent/ongoing responses. A small study comparing boys with ADHD combined type and those who are typically developing, used fMRI data from four visuospatial working memory tasks to classify ADHD with a 92.5% accuracy rate (Hammer et al., 2015). This would need to be replicated in larger samples in order to classify it as a neurobiological marker that could be used in diagnosis. Eye vergence, the movement of both eyes in opposite directions to assist in depth perception, has also shown some evidence as a potential neurobiological marker for ADHD. During attention tasks, eye vergence has been used to differentiate ADHD from controls with a 92% accuracy in children

(Varela Casal et al., 2018), and with 79% accuracy in adults (Jiménez et al., 2021). Additionally, meta-analyses demonstrate that abnormal cortisol and inflammatory biomarker levels could be used as neurobiological markers of ADHD. Morning cortisol levels are lower in children with ADHD compared to those who are typically developing, and the inflammatory biomarker Tumor Necrosis Factor – alpha (TNF-alpha) is also lower in those with ADHD compared to neurotypicals (Chang et al., 2021). Further research needs to be completed on all of the above before any are added to the standard protocol to screen for or confirm ADHD.

C.6. Genetic Factors

ADHD is a complex disorder with no single risk factor or cause. Many environmental and genetic factors contribute to risk, with a multifactorial pattern of inheritance. The relative risk of ADHD in first degree relatives of probands is between 5 and 9, and heritability estimates range from 71% to 90% in twin studies across the globe. This magnitude is similar to that of ASD and schizophrenia (Thapar & Cooper, 2016). Siblings and parents of ADHD patients are two- to eight-fold more affected by ADHD than relatives of healthy individuals. Offspring of parents with ADHD are also observed to have children with more severe forms of ADHD.

Overall, ADHD is more prevalent in males, at ratios of approximately 3:1, although some studies have reported smaller male to female ratios (2.28:1) due to possible underdiagnosing of ADHD cases in females (Ramtekkar et al., 2010).

ADHD is thought to have a polygenic cause, meaning that many genetic variants, each with a small effect, combine together to increase the risk for the disorder. This also means that genetic risk factors of ADHD can also influence lower levels of ADHD symptoms in the general population, that might be below threshold for a full ADHD diagnosis (Demontis et al., 2019; Taylor et al., 2019).

The alteration of genes involved in dopamine functioning have been the most researched in relation to ADHD and hyperkinetic symptoms. The most robust data for gene associations exists for a dopamine transporter gene responsible for dopamine reuptake at the site of neuronal communication and those for dopamine receptors. Studies of variable number tandem repeats (VNTR) in the 3' untranslated region of the dopamine transporter gene have also been shown to predispose individuals to ADHD when combined with prenatal risk factors and environmental insults (Thapar et al., 2012).

Correlations have also been found between risk of ADHD and genes involved in immunological pathways (Drtilkova et al., 2008). Studies have also been completed to explore possible rare genetic variants of ADHD using copy number variants (CNVs) analysis. CNVs were found to contribute to only 0.2% of ADHD heritability, and most that were associated with ADHD were also associated with other psychiatric disorders, including autism and schizophrenia. This suggests a possible overlap in the mechanisms behind these disorders.

MicroRNAs are short, noncoding RNAs responsible for gene regulation after the transcriptional stage. In ADHD, they have been shown to play a role in the regulation of ADHD-associated genes.

It is important to note that although there are several genes and genetic mechanisms involved in ADHD pathology, the results are not always consistent, and much more research needs to be completed.

C.7. Environmental Factors

Epigenetic processes are crucial for normal cellular development and differentiation. They allow for the regulation of gene function through non-mutagenic mechanisms. Epigenetic modification during key developmental periods, prenatally and neonatally, have shown significant

associations with environmental insults present during or around these time periods (Mill & Petronis, 2008). Exposure to all environmental insults identified as risk factors for ADHD are known to occur during these time periods.

Neurons are more prone to epigenetic changes when exposed to an environmental insult when they are undergoing mitosis, or cell proliferation, as they are during key stages of in utero development. Possible epigenetic changes due to exposure to environmental insults, includes histone acetylation, methylation, and phosphorylation; cytosine methylation in CpG islands that can lead to the silencing of a gene and the compaction of chromatin; and RNA-mediated modifications. Changes in methylation patterns have shown significant associations with the severity of ADHD behaviors.

In utero risk-factors associated with ADHD include prenatal exposure to nicotine, alcohol and recreational drugs, in addition to pre- and neo-natal exposure to toxins. These toxins and insults include, but are not limited to, APAP, lead, valproate (Christensen et al., 2019), glucocorticoids, phthalate metabolites, and organophosphates.

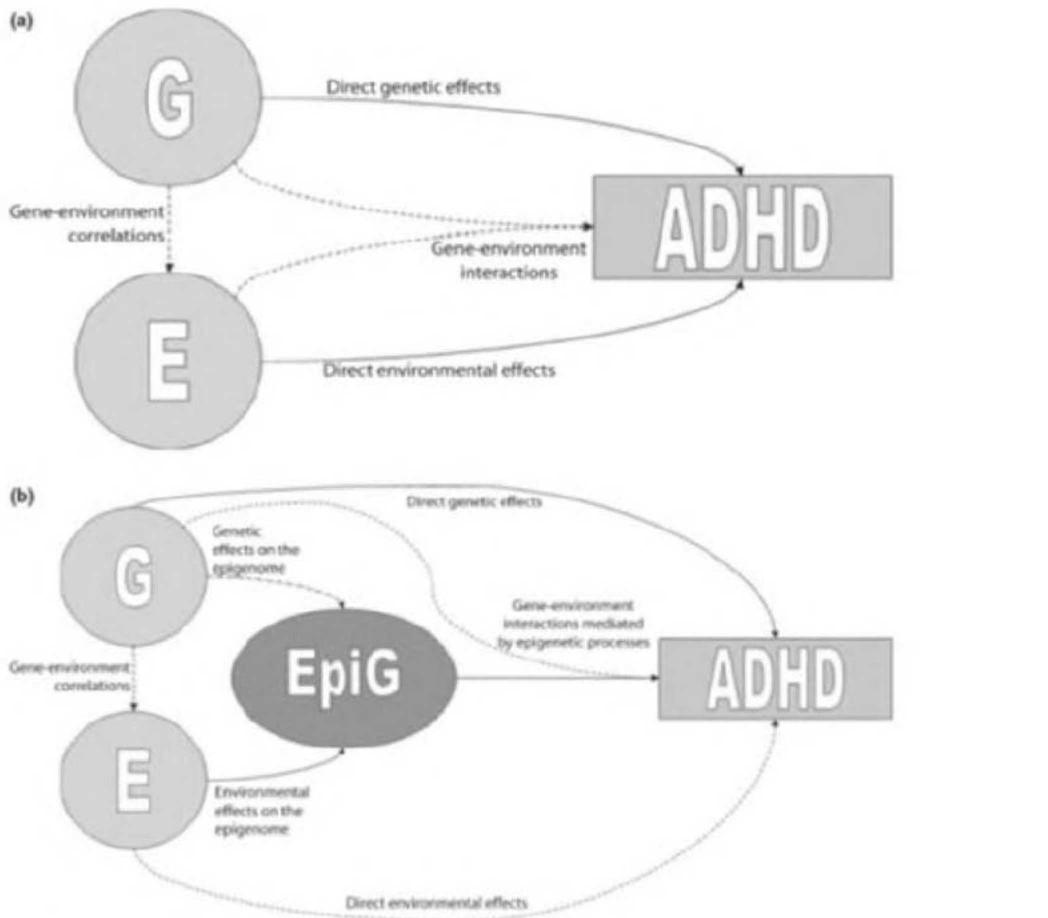


Figure 1 Incorporating epigenetic factors into aetiological models of ADHD. a) Traditional aetiological approaches see susceptibility only in terms of genetic and environmental factors. These factors are generally studied independently, but there is increasing emphasis on gene–environment interactions, whereby the phenotypic effect of genotype is mediated by the environment. b) The epigenetic theory of complex disease includes a third set of aetiological factors, namely those that impact upon the epigenome. Epigenetic dysfunction, mediated via processes such as DNA methylation and histone modifications, may act directly but are also likely to interact with both genetic and environmental factors. Increasing evidence suggests that the phenotypic effects of environmental factors, especially during key developmental periods, may be mediated by epigenetic processes.

(Mill and Petronis, 2008).

An umbrella review of meta-analyses (J. H. Kim et al., 2020) that systematically and quantitatively collected and assessed the hierarchy of evidence for potential environmental risk factors of ADHD found nine associations with high credibility. Five of those risk factors were graded as Class I (convincing evidence): maternal APAP exposure during pregnancy, childhood eczema, hypertensive disorder during pregnancy, preeclampsia, and maternal metabolic syndrome. The remaining four risk factors were regarded as Class II (highly suggestive evidence): maternal

smoking during pregnancy, maternal pre-pregnancy overweight, childhood asthma, and serum vitamin D levels. Maternal APAP exposure during pregnancy and maternal metabolic syndrome are transdiagnostic risk factors, as they were the most robust environmental risk factors for both ADHD and ASD. The largest overall indicator of an environmental insult prenatally is low birth weight, which is also one of the strongest environmental predictors of ADHD. Low birth weight is an early signal of a disruption to the in utero environment, that will have most likely impacted in utero brain development and will lead to symptoms of a neurodevelopmental disorder, such as ADHD or ASD (J. H. Kim et al., 2020; J. Y. Kim et al., 2019). Additional environmental correlates of ADHD during pregnancy and birth include the presence of maternal stress, maternal hypertension, preeclampsia, maternal obesity, maternal infection, and markers of adversity, including poverty and low socioeconomic status. These environmental insults significantly impact the prenatal environment in part due to mediation by epigenetic processes.

The association between this altered prenatal environment and ADHD has been proven in both human epidemiological studies and animal studies (Mill & Petronis, 2008). Some evidence suggests that hyperactive-impulsive symptoms are more likely due to biological risk factors, while inattentive symptoms are more influenced by psychosocial risk factors (Freitag et al., 2012). Some researchers split prenatal risk factors into three categories: chemical exposure during pregnancy, pregnancy or birth complications, and maternal health status. Chemical exposure during pregnancy includes exposure to maternal smoking, exposure to alcohol, polyunsaturated omega-3 fatty acid deficiency, exposure to polychlorinated biphenyls, exposure to APAP, exposure to valproic acid, lead exposure, and pesticide exposure. Pregnancy or birth complications include maternal age, prematurity, low birth weight, and gestational problems. Maternal health status includes maternal

obesity and maternal stress, and possibly maternal anemia during early pregnancy (Faraone et al., 2021; Kian et al., 2022).

C.8. Major Hypotheses on Pathophysiology

Current models of ADHD suggest the main pathophysiological processes involve dopaminergic and noradrenergic neurotransmission impairments, in addition to reduced brain function and volume in key brain areas involved in cognitive processing, attention, motor planning and processing speed, including the prefrontal cortex, caudate and cerebellum. Individuals with ADHD have reduced neural activity and smaller volumes in these areas, in addition to slower maturation of the prefrontal cortex. The prefrontal cortex, caudate and cerebellum are interconnected by a network of neurons, and together regulate attention, thoughts, emotions, behaviors and actions. Network activity between these areas is mostly maintained by dopamine and norepinephrine. Deficits in prefrontal and associated circuit dopaminergic and noradrenergic functioning were initially considered as major aspects of ADHD's pathophysiology, because of the effects of stimulants, which increase synaptic availability of dopamine and norepinephrine, in improving symptoms.

The role of these neurotransmitters has since been further supported by animal model, electrophysiological, and neuroimaging studies. Lesions in dopamine pathways create symptoms of ADHD in animal models. Additionally, individuals with ADHD have alterations in dopamine receptor density in several brain regions compared to neurotypical controls. Further supporting the role of dopaminergic pathways in ADHD pathophysiology are polymorphisms of genes that encode for dopamine receptors (DRD4, DRD 5, DAT1). There is conflict in the literature about the functionality of the dopaminergic and noradrenergic systems in ADHD, with some research positing that these systems are hyperactive in ADHD, and others stating they are

hypoactive/underactive. Recently, these hypotheses have been tied together with the knowledge that dopamine and norepinephrine exhibit inverted U-shaped dose-response curves, where either extreme is problematic. Each area of the brain may access their own supplies of dopamine and norepinephrine, with different ratios available/required for optimal functioning, and any disruption in the DA/NE availability resulting in ADHD symptoms (Sharma and Couture, 2014; Biederman & Faraone, 2005; Purper-Ouakil et al., 2011).

Three related neural circuits are proposed to be involved in the complex behavioral and cognitive symptom profile of ADHD, (1) frontostriatal circuitry; (2) frontocerebellar circuitry; and (3) frontoamygdala circuitry. Frontostriatal and frontocerebellar circuitry are both involved in cognitive control, have dopamine as a critical neuromodulator, and receive noradrenergic projections. Additionally, the cerebellum and basal ganglia both project to the prefrontal cortex via the thalamus. The inhibitory neurotransmitter GABA is primary in the basal ganglia and cerebellum, while the excitatory neurotransmitter glutamate is primary in the prefrontal cortex and the thalamus. Frontostriatal circuitry is key in detecting unpredicted rewarding or novel events, while the frontocerebellar circuitry is responsible for alerting, monitoring and detecting violations in the timing of events. Together, these systems work to monitor the environment and alter behavior in the appropriate context, learning from environmental inputs over the course of child development. They provide neural mechanisms for learning about structure in the environment, and detecting violations in these environmental predictions, which are then integrated with goals represented prefrontally in working memory. Overall, these circuits offer top-down control of behavior, and the ability to alter behavior in response to contextual changes. In the case of a child with ADHD, who is starting off with frontostriatal and frontocerebellar deficits, learning how to

accurately predict what will happen in a given setting, and adapt behaviors to fit the appropriate context will take longer than in a neurotypical child.

For example, a child with ADHD is not able to identify temporally-linked expected events (i.e., sound of a bell means sit down and work), and without knowledge of this temporal association, they are unable to detect when they are violating the norms of the expected event (i.e., continuing to talk after the bell rings), and finally, their lack of inhibitory control prevents them from adjusting their behaviors in a timely manner to fit the expected norm (i.e., stop talking and pay attention to the teacher). Thus, even if the child knows the correct behavior in certain situations, the maladaptive behavior continues due to slower processing, and lack of cognitive and inhibitory control. They would also exhibit poor sustained attention to multistep or complex tasks, slow response in contexts that require a rapid decision, difficulty set-shifting, and inefficient responses to changing learning or reward contexts (Nigg & Casey, 2005). Just as in ASD, this involves disruption of the excitatory/inhibitory balance in the brain, as GABAergic, inhibitory projections would be responsible for interrupting the ongoing behavior when needed, and shifting attention to another task; while glutamatergic, excitatory projections, are involved in maintaining relevant information in the working memory (prefrontal cortex). The third important circuit to ADHD pathophysiology in the frontoamygdala circuitry, which is important for affect regulation, motivation and reactive responses. This circuit is key to the emotional dysregulation observed in those with ADHD, including challenges regulating mood, affect and anger, and dysfunctional approach (positive emotional valence/expectation of reward) and avoidance (negative emotional valence/expectation of punishment) processes. This subsequently results in the impulsive and hyperactive symptoms of ADHD.

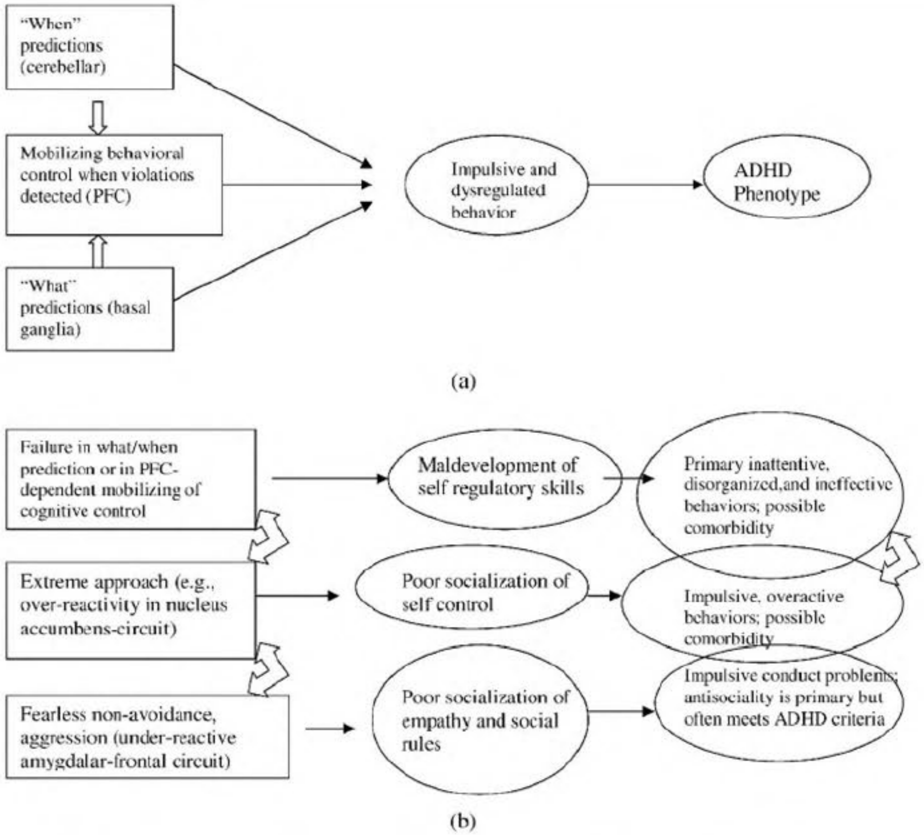


Figure 2. A schematic illustration of the pathways to ADHD symptoms and syndrome: (a) equifinality in ADHD and neuroscience-based hypothesized computational mechanism breakdowns and (b) multiple pathways to related but partially distinct etiological phenotypes in ADHD.

(Nigg and Casey, 2005).

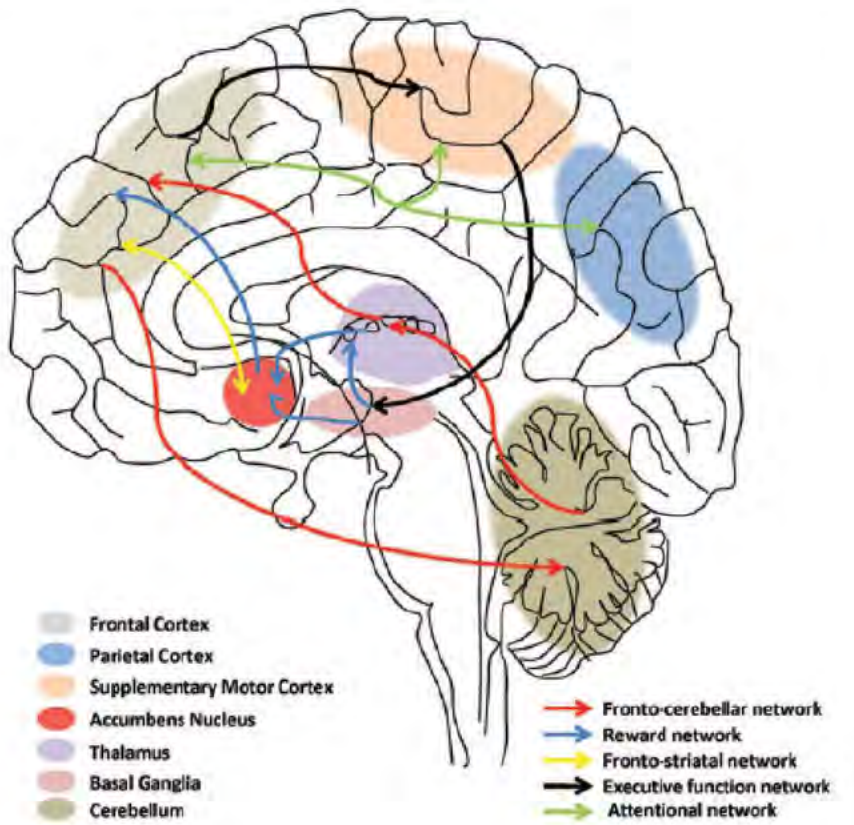


Figure 1. Schematic representation of functional circuits involved in the pathophysiology of ADHD. Here are summarized the attentional network (*green*), the fronto-striatal network (*yellow*), the executive function network (*black*), the fronto-cerebellar network (*red*), and the reward network (*blue*).

(Purper-Ouakil et al., 2011).

In sum, while impairments in prefrontal-striatal circuits result in the inattention and executive functioning deficits observed in ADHD, hyperactivity and impulsivity have been linked to the frontal-limbic system, and reward response and motivation dysfunction. This is also often described as the dual-pathway model of ADHD, as it defines both the cognitive and motivational deficits of ADHD and their respective pathways (Shen et al., 2020), and it is supported by neuroanatomical and neurophysiological studies (Drechsler et al., 2020).

Neuroinflammation may also play a role in the pathophysiology of ADHD (Dunn et al., 2019), as it does for many neurodevelopmental disorders, due to it causing glial activation, increased oxidative stress, neurotransmitter dysfunction, and abnormal neuronal development. Many of the early environmental risk factors for ADHD are also known to increase the inflammatory profile of the in utero environment. This is supported by maternal immune activation (MIA) animal models, in which offspring with aberrant behaviors are born to mothers exposed to prenatal inflammation. MIA animal models are often used to study ASD, but also exhibit behaviors consistent with ADHD, including decreased cognitive flexibility (Bitanihirwe et al., 2010), and difficulties in sustained attention and attentional shifting (Vuillermot et al., 2012). There are also animal models for the hyperactivity symptoms of ADHD. Children with ADHD tend to exhibit this symptom in environments they are comfortable in, as opposed to new situations and places, which is seen in animal models as increase locomotor behavior in the home cage, as opposed to a new stressful setting. Offspring of mothers exposed to poly(I:C) (an infection) during gestation, show increases in locomotor behavior in their home cage (Missig et al., 2018; Miller et al., 2013).

However, there are also some studies that show the opposite behaviors, and more research needs to be done to find an appropriate animal model of hyperactivity. Of note, the results in these models appears to be dependent on gender, age and context. Male offspring locomotor activity appears to be most affected by MIA, which would be consistent with symptom presentation across genders in humans. MIA animal models also display impulsivity, another key feature of ASD. In rodent models this is exhibited by increased locomotion, decreased inhibition, social intrusiveness and aberrant social behaviors. Lastly, MIA animal models also demonstrate impaired working memory and executive functioning deficits, similar to their human counterparts with ADHD. These behavioral changes correlate with structural changes to the brains of animal models, including

decreased overall brain volume and volume of cortical areas associated with ADHD. Exposure to MIA during gestation also alters the function of neurotransmitters associated with ADHD symptoms, including dopaminergic, serotonergic, glutamatergic and GABAergic systems, which is seen in both animal and human studies.

Although the data is preliminary, individuals with ADHD also present with higher rates of atopic immune disorders and have higher serum cytokine levels than their neurotypical peers (Lin et al., 2016; Instanes et al., 2017; Schans et al., 2017; Schmitt et al., 2010; Miyazaki et al., 2017; Anand et al., 2017; Darwish et al., 2018). Preliminary studies also show an association between proinflammatory serum cytokines and ADHD symptom severity (Oades et al., 2010), with lower levels of cytokines present in those with ADHD currently taking methylphenidate.

The endocannabinoid system, and disruptions to its functioning, may also contribute to ADHD symptomatology (Giuffrida et al., 2001). Endocannabinoids play a key neuromodulatory role in dopamine-related pathophysiological responses, like those involved in ADHD (Tzavara et al., 2006). N-4-hydroxyphenyl-arachidonylamide (AM404) is an inhibitor of endocannabinoid reuptake, increases circulating anandamide levels and inhibits motor activity, and reduces behavioral responses to dopamine agonists. In juvenile spontaneously hypertensive (SHR) rats, an animal model with hyperactivity and attention deficits, the administration of AM404 normalizes these behaviors (Beltramo et al., 2000). This suggests that inhibitors of endocannabinoid inactivation may alleviate dopamine dysfunction symptoms. It also confirms the role of the endocannabinoid system in psychomotor activity, and in ADHD pathophysiology. Genetic studies of polymorphisms related to ADHD symptomatology have also confirmed the role of the endocannabinoid system. A polymorphism in the endocannabinoid degrading enzyme FAAH (FAAH rs2295633) was found to be significantly associated with ADHD in a study of children

with ADHD compared to healthy controls (Ahmadalipour et al., 2020). The FAAH gene is involved in reward and addiction, via its effects on the endocannabinoid and dopaminergic pathways. It has a direct effect on the degradation of endocannabinoids in the brain and is linked to arousability. Increased levels of FAAH are linked to decreased levels of endocannabinoids, and if FAAH is inhibited, the subsequent increase in endocannabinoid level is shown to reduce anxiety levels and increase aversive memory extinction in animal models.

Oxidative stress may also contribute to the pathophysiology of ADHD (Joseph et al., 2015). A meta-analysis of six studies examined the association of ADHD and oxidative stress and antioxidant status in medication naïve patients with ADHD and neurotypical controls. Although the association between ADHD and antioxidant status was not significant, the association between oxidative stress and ADHD could not be accounted for by publication bias, sample characteristics, study design features, or unusual results. Although preliminary, these results suggest that individuals with ADHD have an insufficient response to oxidative stress, resulting in oxidative damage. This is demonstrated by the increased ratio of oxidative to antioxidative status in the ADHD group. Oxidative damage can include, but is not limited to, altered protein structures, epigenetic changes, and mitochondria dysfunction.

C.9. Pharmacological and Psychological Treatments

ADHD is one of the most commonly diagnosed and treated childhood neurodevelopmental/psychiatric disorder, with wide variations in medical practice across the globe. Psychological interventions include behavioral therapies, cognitive training, and neurofeedback. Behavioral interventions have been extensively studied and form a core part of intervention guidelines for ADHD. Behavioral therapies in combination with stimulants are superior to the use of only medication, but stimulants continue to be superior to behavioral therapy,

cognitive therapy, and non-stimulants (Catalá-López et al., 2017). Pharmacological interventions for ADHD include not only stimulants, the most supported and evidence-based treatment, but also non-stimulants, antidepressants, antipsychotics, and other unlicensed drugs. The treatment process can be very complex.

D. The Interrelationship of ASD and ADHD

There is significant overlap and co-morbidity between the symptoms of ASD and ADHD, and there may be overlap in the underlying biology that accounts common features that supersede traditional diagnostic categories. The most recent edition of the DSM allows for a dual diagnosis of ASD and ADHD, recognizing the significant overlap of these disorders and that they frequently co-occur. The diagnostic assessments often used to identify these disorders also overlap. I have included a list of common assessments at **Appendix 3**.

Between 30% and 75% of children with a diagnosis of ASD present with symptoms of ADHD, and 20% to 60% of those with ADHD present with the social difficulties common to ASD (Grzadzinski et al., 2016). The variance in prevalence rates can be explained by the only recent allowance to give dual-diagnoses in the DSM-5.

Children with ADHD regularly present with social dysfunction, and these social communication deficits are similar to those present in ASD. Restricted and repetitive behaviors, common to ASD, are also found in those with ADHD. ASD symptoms appear to be related to the severity of symptom presentation in those with ADHD, with those with more severe behavioral difficulties, oppositional behaviors and depression, being more likely to have symptoms of ASD. (Charman et al., 2007; Clark et al., 1999; Cooper et al., 2014; Frazier et al., 2008; Greene et al., 1996; Grzadzinski et al., 2011; Hus et al., 2007; Hus & Lord, 2013; Luteijn et al., 2000; Martin et al., 2014; Marton et al., 2009; Mulligan et al., 2009; Nijmeijer et al., 2009; Reiersen et al., 2007).

ADHD and ASD are noted to share similar endophenotypes, including difficulties with emotion regulation, externalizing behaviors, and social communication deficits. This could account for the severity of impairment present in those with ASD and ADHD, as children with both diagnoses are noted to have greater cognitive and social impairments and higher rates of externalizing and internalizing behaviors. Children with ASD and ADHD perform worse on continuous performance tasks, including presenting with more attention deficits, higher activity levels and greater impulsivity than children with ASD alone (Stevanovic et al., 2022).

There is considerable genetic, neuropsychological and clinical overlap between ASD and ADHD, and the DSM-5 now allows for the two to be diagnosed concurrently. Studies have shown that between 22% and 83% of children with ASD have symptoms that meet criteria for ADHD (Sokolova et al., 2017). Children with ADHD are also shown to have high rates of ASD symptomatology. A recent study showed that 12.5% of children currently diagnosed with ADHD also had an ASD diagnosis (Zablotsky et al., 2017). ASD and ADHD share about 50% to 72% of contributing genetic factors, and have similar deficits in motor speed, social cognition, impulsivity and executive function (Sokolova et al., 2017). Inattention and decreased attentional switching capacity also overlap and may be linked to similar biological pathways. In clinical practice it may be difficult to determine if the impulsivity of ADHD is responsible for the social communication problems of ASD, or vice versa, and if the repetitive behaviors of ASD are mistaken for hyperactivity of ADHD, or vice versa. Thus the two diagnoses may be difficult to tease apart (Sokolova et al., 2017; Zablotsky et al., 2017).

There is significant between-group overlap and within-group heterogeneity, for complex disorders such as ASD and ADHD, as well as with other neurodevelopmental disorders. Clinicians must take a transdiagnostic approach in their assessments not only in children, but across the

lifespan (Kushki et al., 2019). Without this approach, the similarities in behavioral profiles between these disorders could lead to challenges in both the diagnosis and intervention efforts, particularly as individuals can have comorbid diagnoses of these disorders, with their own unique symptom profiles.

IV. NEURODEVELOPMENT AND APAP

A. Background of Acetaminophen

Acetaminophen, also known as paracetamol, is a widely used ingredient found in many over-the-counter (OTC) and prescription medicines for relieving fever and pain. The most well-known brand name for APAP is Tylenol®, but it is available under various other brand names and formulations. APAP is often combined with other active ingredients to treat conditions like allergies, coughs, colds, flu, and sleeplessness. In prescription medications, it is used alongside other ingredients to manage moderate to severe pain. It comes in different forms such as syrups, tablets, caplets, capsules, effervescent tablets, injections, suppositories, and more.

APAP was first developed by pharmacologist Joseph von Mering in 1893 through reacting p-nitrophenol with tin and glacial acetic acid. (Ayoub, 2021). This newly synthesized compound was known to have analgesic (pain-relieving) and antipyretic (fever-reducing) properties. APAP was approved by the U.S. Food and Drug Administration (FDA) for prescription use under the brand name Tylenol®, and in 1955, it became available for nonprescription OTC use in the United States. Since its approval by the FDA, acetaminophen has become one of the most widely used OTC non-narcotic analgesic agents for treating mild to moderate pain and fever (Ayoub, 2021).

When ingested, APAP is quickly absorbed from the gastrointestinal tract and reaches peak concentrations in the blood within 1 to 2 hours. It is primarily metabolized in the liver and eliminated mainly through the urine. The exact mechanism of action of APAP is not completely understood, but it is believed to involve several processes in the body. The primary mechanism is

thought to be the inhibition of prostaglandin synthesis in the central nervous system (CNS) (Bührer et al., 2021). Prostaglandins are chemical messengers that play a role in inflammation, pain, and fever that are produced in response to injury or illness and contribute to the sensitization of pain receptors and the generation of fever. APAP is believed to inhibit the activity of the enzyme cyclooxygenase (COX), particularly the COX-2 isoform, which is responsible for the synthesis of prostaglandins. By inhibiting COX-2, APAP reduces the production of prostaglandins in the CNS. This, in turn, helps to alleviate pain and reduce fever. Unlike nonsteroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen or aspirin, APAP has limited effect on COX-1, which is involved in maintaining the protective functions of the stomach lining and platelet aggregation. While APAP has analgesic (pain-relieving) and antipyretic (fever-reducing) effects, it has limited anti-inflammatory properties relative to NSAIDs. Its action primarily focuses on the CNS and does not significantly impact peripheral tissues (Anderson, 2008; Ghanem, 2016). APAP's analgesic effect may be weaker than previously thought (Moore, et al., 2014). For instance, Moore et al. concluded that ibuprofen produced more effective pain relief in patients than APAP at standard doses in different painful conditions (Moore et al., 2014).

Consumers may unintentionally misuse non-prescription medications like APAP, including taking more than the recommended dose according to the product label (Wolf, et al., 2012). This may be due to the consumer's misconception that APAP is harmless (Wolf, et al., 2012). Additionally, access to higher doses of APAP may increase the risk of unintentional misuse and lead to up-dosing. For instance, Martinez-De La Torre, et al. found that after a 1,000 mg tablet of APAP was introduced, the number of accidental poisoning cases due to APAP increased (Martinez-De La Torre, et al., 2020).

A.1. Questionable Safety Profile For Prenatal Use of Acetaminophen

In my clinical practice, I have experience engaging in a risk/benefit analysis when prescribing and/or recommending medications to pregnant women. In that role, it is important to engage in a meaningful conversation with the pregnant patient discussing the benefits as well as the potential risks of taking a medication while pregnant.

To undertake these risk–benefit calculations and recommendations for my pregnant patients, it is necessary that a drug manufacturer or seller disclose all potential risks and adverse pharmacological effects associated with taking the drug. Full disclosure of potential risks, adverse effects, or risk rating allows me to effectively inform my patients of the risks and benefits associated with the drug and to provide accurate recommendations. This is critical because there is an asymmetry of information between the manufacturer and healthcare providers, and we rely on the manufacturers to provide truthful information. If manufacturers and sellers do not provide comprehensive and adequate information about the potential risks or adverse effects of a particular drug, including a risk rating, healthcare professionals are unable to make definitive, evidence-based clinical decisions and recommendations for their patients' treatment and care.

APAP has been sold and represented as safe for ingestion during pregnancy for decades, and Johnson & Johnson has represented to clinicians, such as myself, that it is a "Category B" pregnancy drug category. From 1979 through 2015, the FDA required prescription drugs to be classified into one of five categories to indicate risk of use while pregnant, and such classification was required to be included on the product label (Pernia & DeMaagd, 2016).

FDA Pregnancy Drug Categories	
Category A	No risk shown in human studies. Adequate and well-controlled studies have failed to demonstrate a risk to the fetus in the first trimester of pregnancy (and there is no evidence of risk in later trimesters).
Category B	No risk in animal studies (there are no adequate studies in humans, but animal studies did not demonstrate a risk to the fetus). Animal reproduction studies have failed to demonstrate a risk to the fetus and there are no adequate and well-controlled studies in pregnant women.
Category C	Risk cannot be ruled out. Animal reproduction studies have shown an adverse effect on the fetus and there are no adequate and well-controlled studies in humans, but potential benefits may warrant use of the drug in pregnant women despite potential risks.
Category D	Evidence of risk (studies in pregnant women have demonstrated a risk to the fetus; potential benefits of the drug may outweigh the risks). There is positive evidence of human fetal risk based on adverse reaction data from investigational or marketing experience or studies in humans, but potential benefits may warrant use of the drug in pregnant women despite potential risks.
Category X	Contraindicated (studies in pregnant women have demonstrated a risk to the fetus, and/or human or animal studies have shown fetal abnormalities; risks of the drug outweigh the potential benefits). Studies in animals or humans have demonstrated fetal abnormalities and/or there is positive evidence of human fetal risk based on adverse reaction data from investigational or marketing experience, and the risks involved in use of the drug in pregnant women clearly outweigh potential benefits.

Table 1. Content and Format of Labeling for Human Prescription Drug and Biological Products; Requirements for Pregnancy and Lactation Labeling (Federal Register/Vol. 73, No. 104/Thursday, May 29, 2008).

Based on some internal Johnson & Johnson documents, it seems there is some question as to whether Tylenol should be classified as "Category B" or whether it ever was (Exhibit 25 to Deposition of [redacted]; Exhibit 27 to Deposition of [redacted]). Johnson & Johnson's Professional Product Information manual for Tylenol, however, which was drafted to be used by healthcare professionals such as myself, states that "Acetaminophen is routinely used during all stages of pregnancy and is categorized with FDA Pregnancy Risk Factor Rating of B (i.e., animal

reproduction studies have failed to demonstrate a risk to the fetus and there are no adequate and well controlled studies in pregnant women or animals that have shown adverse effect)" (Exhibit 9 to Deposition of [redacted]). In support of that statement, the Professional Product Information cites to an outdated version of a textbook (Briggs et al., 2005). The most recent version of the Briggs textbook, however, states that "use of the [APAP] for several weeks or longer has been associated with cryptorchism, decreased IQ, ADHD, and other problems in neurodevelopment," and that "[a]lthough originally thought not to cause embryo-fetal harm, this assessment must change because of recent data" (Briggs et al., 2021).

In addition, I have identified two other products containing APAP that acknowledge fetotoxicity studies concerning oral administration of acetaminophen in pregnant rats, studies which would render APAP a "Category C" under the FDA's category system. Ofirmev is a pure APAP injection, and that label states that animal studies in pregnant rats that received oral APAP "showed evidence of fetotoxicity" and that it should only be given to a pregnant woman only if "clearly needed" (Exhibit 9 to Deposition of [redacted]). The Maternal Health team for the FDA ultimately designated Ofirmev as a Category C drug, meaning risk cannot be ruled out as animal reproductions studies have shown an adverse effect on the fetus and there are no adequate well controlled studies on humans. (Dept of Health Ofirmev Review, 2010).

Second, Ultracet is a pharmaceutical product manufactured by Janssen, a subsidiary of Johnson & Johnson, that contains APAP and tramadol. It is a combination of acetaminophen and tramadol. The Ultracet label has a section that solely focuses on APAP that says, "acetaminophen approximately 1.3 times the maximum human daily dose (MRHD) showed evidence of fetotoxicity and increases in bone variations in the fetuses" (Ultracet Label).

A.2. Brain Development in Utero

The development of the CNS and brain in utero is a complex, highly orchestrated process that involves the proliferation and migration of cells, the formation of neural circuits, and the establishment of synaptic connections. There are multiple stages to human brain development, including neurulation, neurogenesis/proliferation, cell migration, differentiation, synaptogenesis, and myelination.

The nervous system originates from the ectodermal layer of the germ disc (the outermost layer of the embryo), which is the flat surface adjacent to the amnion (the innermost membrane that encloses the embryo) (Rodier, 1995). Along the midline of the disc, a groove emerges, and the folds on each side come into contact and merge. This fusion gives rise to a tube structure that remains open at the head and tail ends of the developing embryo, then the neural tube closes at the head and tail ends first, and then in the middle. Once the neural tube is closed, it forms the brain and spinal cord. This process is called neurulation. The process of tube formation initiates in the cervical region and progressively extends towards the head and tail of the embryo. Typically, the closure of the tube is observed between approximately three to four weeks after conception.

The neurogenesis/proliferation stage begins during the first month of gestation in humans and involves the production of neurons (nerve cells), which begins even before the neural tube is closed (Rodier, 1995; Rice & Barone, 2000). The CNS is made of dozens of different types of neurons (Rodier, 1995). Neural stem cells divide and differentiate into precursor cells, which then develop into neurons. Neurogenesis primarily occurs in specific regions of the brain, such as the ventricular zone. During this stage, the overproduction of neurons is balanced out by a process of programmed cell death called apoptosis.

After neurogenesis, newly formed neurons migrate from their site of origin to their final destinations in the brain (Rodier, 1995). This process is critical for establishing the correct architecture and connectivity of the developing brain. Neurons migrate along specialized pathways, guided by chemical signals and radial glial cells. The majority of cell migration takes place during the early stages of gestation when the distances within the brain are relatively short. However, the extensive migrations of small cells in regions like the cerebral cortex, hippocampus, and cerebellum persist for several months following birth.

Once neurons reach their proper locations through migration, it typically follows one of two paths: differentiation into a fully developed neuron with axons and dendrites, or elimination through apoptosis (Rodier, 1995). Axon development relies on growth cones, small structures at the axon's edge, which guide growth towards specific targets while avoiding others. Early dendrites emerge as thick strands with few spines (small protrusions) extending from the cell body. As dendrites mature, the number and density of spines increase, enhancing the likelihood of dendrites establishing connections with neighboring axons. These connections between dendrites and axons form the basis for synaptic connections between neurons, which are essential for proper brain function.

To fulfill their role as signal transmitters, neurons must establish connections, which necessitates the development of specialized structures on the surfaces of both sending and receiving neurons (Rodier, 1995). The site of contact where these structures meet is known as a synapse. It is at the synapse that neurons interact and facilitate the transmission of signals to achieve mature functionality. During synaptogenesis, synapses begin to form. Axon terminals establish contact with dendrites or other target cells, thereby creating functional connections. Synaptic formation and refinement occur in a highly dynamic and activity-dependent manner.

Myelination involves the production of a fatty substance called myelin, which wraps around axons. Myelin acts as an insulating sheath, enhancing the speed and efficiency of nerve signal transmission (Rodier, 1995). Myelination progresses gradually throughout childhood and continues into adolescence.

Neural development extends from gestation through adolescence (Rice & Barone, 2000). Critically, the formation of the blood-brain barrier is a gradual process that commences during fetal development but does not reach completion approximately six months after birth in humans.

A.3. Susceptibility of the Developing Brain to Toxicity

The developing brain is highly sensitive to environmental influences. As noted above, the blood-brain barrier, which protects the brain, is not fully formed during fetal development. As a result, the developing brain is more permeable to chemicals and medications compared to the mature brain (Rodier, 1995). Additionally, the brain undergoes rapid growth during the second trimester of pregnancy, followed by a series of critical processes (including neurulation, neurogenesis/proliferation, migration, differentiation, and synaptic pruning) throughout early childhood (Rodier, 1995).

This prolonged development makes the brain more vulnerable to exposures and insults than other organs in the body (Rodier, 1995; Rice & Barone, 2000). Additionally, the brain is composed of various types of neurons and other cells like oligodendrocytes, astrocytes, and microglia, each with its own distinct growth phase and potential susceptibility to toxicity. The vulnerability of the developing brain to the effects of chemicals during pregnancy is widely recognized as a biological fact in the scientific community. Factors such as the incomplete development of the blood-brain barrier and the ongoing growth, differentiation, and pruning of neurons contribute to the heightened vulnerability of the developing brain to exposures.

Due to the intricate nature of the brain, which consists of numerous interconnected circuits, the establishment of fully mature neural systems involves a greater number of developmental processes compared to other tissues. Consequently, there are increased opportunities for potential harm or injury. The most significant aspect of the CNS in relation to developmental mishaps, however, is the prolonged duration over which CNS development takes place.

B. Interrelationship Between Genetics and/or Fever/Infection and APAP

The Maternal Immune Activation (MIA) hypothesis discusses the relationship between infection or fever during pregnancy and an increased risk for ASD, ADHD, and other neurodevelopmental disorders in offspring. Viral infection during the first trimester resulting in hospitalization, and bacterial infection in the second trimester are associated with a diagnosis of ASD in offspring (Atladóttir et al., 2010). Additionally, self-reported influenza during pregnancy was associated with a twofold increased risk of infantile autism; while febrile episodes lasting more than 7 days are associated with a threefold increase in risk (Atladóttir et al., 2012). It is noted that these maternal infections and febrile episodes may increase the mother's exposure to APAP and other NSAIDs, and that the two hits of infection/fever during pregnancy and fetal exposure to APAP could further exacerbate fetal insult, increasing the risk for the development of neurodevelopmental disorders, including ASD and ADHD. The majority of studies reviewed examining the association between prenatal APAP exposure and development of ASD/ADHD symptoms do control for maternal infection and fever, and the association is still present.

The effect of MIA on offspring neurodevelopment and behavior has been demonstrated in multiple animal models using a variety of immune system activators, including the influenza virus, the viral mimetic polyinosinic-polycytidylic acid (polyI:C), interleukin-6, and the bacterial endotoxin lipopolysaccharide (LPS). Early prenatal stress can also act as an immune system activator, and it has been shown to produce changes in gene expression patterns, including the up-

regulation of immune-related genes of proinflammatory cytokines and chemokines (Bronson & Bale, 2014). Notably, the timing of the MIA is critical to the subsequent effect on the fetus, as early first trimester and second trimester insults causing the most significant insult (Garay et al., 2013). Alterations in brain cytokine levels are found throughout the lifespan of MIA offspring. In both the frontal cortex and cingulate cortex, proinflammatory and anti-inflammatory cytokines are elevated in the early postnatal period, lower than controls during early adolescence, and elevated again in the adult brain. The hippocampus also has alterations of cytokine levels at each age level, but these are distinct compared to other brain areas. There also appears to be a widespread decrease in cytokine levels during critical periods of synaptogenesis and plasticity in MIA offspring compared to controls. This is in contrast to the expected pro-inflammatory phenotype and may indicate an alternative pathway for cytokines to impact brain connectivity and subsequent behavior in ASD. Of note, brain cytokine levels are different than serum cytokine levels, indicating that the levels in brain tissue are not contaminated by serum. Additionally, ASD offspring have no changes in blood-brain barrier permeability, immune cell infiltration or microglial density (Garay et al., 2013; Patterson, 2012; Ito et al., 2010; Shi et al., 2009; Arrode-Brusés & Brusés, 2012).

Offspring of MIA mice exhibit the three core features of ASD, including deficits in communication, sociability, and the presence of repetitive and stereotyped behavior. Mirroring the results observed in animal models, large epidemiological studies have found significant associations between maternal viral infections during the first trimester of pregnancy and increased autism risk in their children (Atladóttir et al., 2012). Maternal infections from a variety of microorganisms, including influenza, varicella, and rubella, have been associated with an increased risk for developing ASD, demonstrating that it is not the particular pathogen that is responsible, but rather the generalized activation of the maternal immune system during pregnancy

(Atladóttir et al., 2012; Zerbo et al., 2014). Additionally, prolonged febrile episodes and antibiotic use has been linked to an increased risk for infantile ASD (Atladóttir et al., 2012). A study completed by the Childhood Autism Risks from Genetics and Environment (CHARGE) group evaluated both maternal influenza and maternal febrile episodes during pregnancy and its relationship with ASD risk. The development of ASD or developmental delays was significantly associated with maternal febrile episodes but not with maternal influenza during pregnancy. Additionally, the ASD risk due to fever was attenuated when mothers reported taking antipyretic medications (Zerbo et al., 2013). These insults are comparable to a study completed in Denmark that showed an association between admission to the hospital in the first trimester for maternal viral infection and the second trimester for maternal bacterial infection with a diagnosis of ASD in offspring (Atladóttir et al., 2010). However, not all infections have the same result, as some viral infections (i.e., influenza) appear to be heavily correlated with increased ASD risk, while urinary tract infections, upper respiratory infections, and cystitis do not appear to be risk factors. Although there is significant data from mouse models on the detriment of MIA, further work is needed in humans to expand on the epidemiological research mentioned above.

C. Role of Animal Studies

Animal models provide critical information to epidemiological, clinical, and biomedical research by offering a controlled experimental setting to study diseases/disorders and their mechanisms. Animal models mimic aspects of biologic processes or diseases found in humans and have an important role in studying toxicity, neurodevelopment, pharmacokinetics, and in completing the pre-clinical testing often needed for drug development. By mimicking human conditions and exposures in animals, researchers can study the progression, etiology, and pathophysiology of diseases/disorders. This understanding is crucial for identifying risk factors, developing prevention strategies, and designing targeted interventions.

Animal models are extensively employed in investigating the neurobiology of psychiatric disorders and understanding how current and new psychotropic medications work (Nestler et al., 2002; Desbonnet et al., 2012). Using animal models, neuroscience has gained valuable insights, although it should be acknowledged that these models are limited to simulating specific aspects of psychiatric syndromes rather than providing an entirely accurate and comprehensive representation of the conditions (Neumann et al., 2011; Stephens et al., 2013).

Animal models also provide researchers with a controlled experimental environment where various factors can be manipulated and studied. This control allows for the isolation of specific variables, which helps establish causal relationships between exposures and health outcomes. Animal models also enable the systematic testing of hypotheses and the evaluation of potential interventions in a controlled setting.

Animal models offer certain practical advantages in terms of feasibility and logistics by allowing for longitudinal studies, precise follow-up, and standardized data collection. More generally, animal models can be used to study rare diseases, complex interactions, and long-term effects that may be difficult to assess in human populations due to sample size limitations or ethical constraints. In the case of studying the association between maternal APAP use during pregnancy and the association with neurodevelopmental disorders like ASD and ADHD, for instance, it is unethical to perform randomized controlled trials (RCTs) in humans. However, multiple meaningful animal studies have been performed to study the association between environmental exposures during pregnancy and the development of neurodevelopmental disorders in offspring. These animal studies allow us to analyze the relationship between fetal exposures and the development of symptom domains associated with both ASD and ADHD, including hyperactivity, social deficits, repetitive behaviors, and irritability. For example, the valproate mouse model is an

animal model of autism that reliably demonstrates social deficits, repetitive behaviors, and other symptom domains of the autism spectrum (Rodier, 1996; Nicolini & Fahnestock, 2019). Drugs developed for the treatment of autism are often screened in this and other animal models to determine whether they should be pursued in clinical trials in humans. For these reasons, animal studies have been used for decades to investigate causation in ASD and ADHD (Rodier, 1996).

As further explained in the expert report of Dr. Pearson, animal models serve as a bridge between basic research and clinical applications. Findings from animal studies can inform and guide human epidemiological studies, clinical trials, and the development of preventive or therapeutic strategies. Animal models provide insights into potential mechanisms, biomarkers, and treatment targets, aiding in the translation of research findings into practical applications for human health. Further, animal models are instrumental in the validation and preclinical testing of potential therapies, vaccines, or interventions before they are tested in humans. Animal studies help assess safety, efficacy, dosage, and potential adverse effects, providing critical information for the development and refinement of medical interventions. In this case, animal studies allow researchers to look at the presentation of neurodevelopmental symptoms over the lifespan and its relationship to various fetal exposures independent of other confounders.

D. Mechanism of Action & Biological Plausibility

As more thoroughly detailed above, fetal brain development is a complex, tightly regulated process that, if disrupted, can lead to long-lasting effects on the brain. As explained below, APAP can impact fetal brain development and lead to neurodevelopmental disorders in offspring through multiple mechanisms of action.

The causal association between fetal APAP exposure and neurodevelopmental disorders, specifically ASD and ADHD, is biologically plausible based on the known mechanisms of action

discussed below and as discussed in greater detail within the expert reports of Drs. Cabrera and Pearson.

D.1. APAP Can Directly Affect the Fetus

APAP crosses the placenta barrier and has endocrine-disrupting properties that interrupt fetal brain development by interfering with maternal hormones or via neurotoxicity, including the induction of oxidative stress that could result in neuronal death. The placenta, the organ developed during pregnancy, acts as a link between the mother and developing fetus and serves as a barrier between the maternal and fetal circulatory systems (Gude et al., 2004). This separation of circulatory systems protects the fetus from potentially harmful substances within the mother's circulation system, such as drugs, infectious agents, and toxins (Robbins et al., 2012). In some instances, however, a substance may cross the placental barrier and enter the fetal circulatory system. APAP is a small molecule that is known to cross the placenta and enter the fetal circulatory system (Weigand et al., 1984; Thiele et al., 2013; Nitsche et al., 2017). Thus, APAP can reach the fetus and affect its development (Thiele et al., 2013).

D.2. Excess NAPQI Formation

APAP has multiple important metabolic pathways, including glucuronidation and sulfation. APAP is primarily metabolized in the liver through conjugation with glucuronic acid (reduced glutathione or "GSH") and sulfate, which results in the excretion of nontoxic final products (Bauer et al., 2018). These metabolic routes generally yield inactive, non-toxic, final products. Glucuronidation is the primary metabolic pathway in adults, while sulfation is the primary pathway for APAP metabolism until ages 10–12 years. One of the secondary pathways is mediated by cytochrome P-450. However, the remainder is oxidized in the liver through several CYP450 isoforms (CYP2E1, CYP1A2, CYP3A4, and CYP2A6) to produce the metabolite N-acetyl-

benzoquinone imine (NAPQI). NAPQI is generally normalized by combining with GSH, which is converted to non-toxic metabolites and excreted (Toussaint et al., 2010).

If concentrations of NAPQI exceed the available GSH, NAPQI initiates a mitochondrial cascade that leads to accumulation of reactive oxygen species and cell death (Jaeschke, 2021; Jaeschke, 2020). Supratherapeutic doses of APAP may lead to excessive NAPQI formation and result in acute liver failure (ALF) and hepatic encephalopathy (Jetten et al., 2016; Zhao & Pickering, 2011; Butterworth, 2011). Even therapeutic doses have been found to be associated with hepatotoxicity. When cellular GSH is depleted, NAPQI binds to cellular proteins including mitochondrial proteins. This can reduce the body's ability to detoxify, which can lead to immune system activation and oxidative stress. It has been shown that brain cells can metabolize APAP to produce NAPQI (Ghanem et al., 2016; Upadhya 2000). Ghanem's finding that NAPQI is generated in the brain reveals that cortical inflammation and oxidative stress may contribute to abnormal neurodevelopment. Male mice treated with APAP have shown greater GSH depletion, and altered GSH homeostasis has been observed in children with ASD and their mothers (Oken et al., 2007; Williams et al., 2007; Bowers et al., 2011). Women who used APAP in the third trimester had an increased risk of preeclampsia in the Danish Birth Cohort (Rebordosa et al., 2010). Additionally, preeclampsia has been linked to decreased glutathione levels, which could be further exacerbated by APAP use, and has been linked to an increased risk of having a child with ASD (Mann et al., 2010; Buchmayer et al., 2009). The cytochrome P-450 pathway could also play a role in the reduced sulfation capacity observed in pregnancy, which can activate immune response pathways, including pro-inflammatory cytokine interleukin signaling (Davies et al., 1994; Lee et al., 2009). Similar cytokines are involved in the maternal immune activation hypotheses of ASD pathophysiology. Additionally, therapeutic doses of APAP are also linked to immune activation

and pro-inflammatory cytokine signaling (Jetten et al., 2012; Wright et al., 2007; Deverman & Patterson, 2009). Thus, prenatal exposure to APAP may trigger maternal immune activation pathways, similar to those noted in the MIA pathophysiology of ASD. The mechanism of analgesic action in APAP metabolism involves the deacetylation of APAP in the liver, which subsequently produces p-aminophenol. P-aminophenol is involved in APAP nephrotoxicity and in mouse models produces significant losses of cortical neuron viability (Schultz et al., 2012).

D.3. Oxidative Stress

Oxidative stress is a phenomenon that occurs due to an imbalance between the production of reactive oxygen species (ROS) and the body's ability to reduce their harmful effects by using antioxidants (Manivasagam et al., 2020). The human brain uses the most oxygen of any organ in the body (Liu et al., 2022). The developing human brain is highly susceptible to harm caused by reactive oxygen and reactive nitrogen species, in part due to abundance of unsaturated fatty acids, elevated oxygen consumption, limited presence of antioxidants, significant metal content that facilitates free radical production, and considerable population of delicate immature cells (Liu et al., 2022; Ikonomidou & Kaindl, 2011). The developing fetal brain can be adversely affected by oxidative stress, and prenatal APAP exposure has been shown to increase oxidative stress through multiple mechanisms (Parker et al., 2017). APAP depletes glutathione and other antioxidants that neutralize ROS (Brune et al., 2015). Glutathione is used when APAP is metabolized in the liver, and its resulting depletion means that glutathione is not available for other tissues such as the fetal brain (Brune et al., 2015). The fetal brain is rapidly growing and maturing and requires high energy metabolism, but at the same time, it is very vulnerable to oxidative stress. Oxidative stress can result in cellular damage, disrupted neural connection formation, and abnormal brain function (Ikonomidou et al., 2010). APAP exposure in utero has been shown to increase oxidative stress

markers in the fetal brain and has been associated with adverse neurodevelopmental outcomes or deficits in animal studies (Bauer et al., 2018; Blecharz-Klin et al., 2015, 2016, 2017; Viberg et al., 2014).

D.4. Effects on the Prostaglandin System

APAP's antipyretic, or fever reducing, effect is suggested to be due to its effect on the prostaglandins, which mediate the generation of fever and are involved in neuronal development and synaptic plasticity in the developing brain (Bauer et al., 2018; Mirrasekhian et al., 2018; Dean et al., 2012). APAP's antipyretic properties are attributed to its ability to inhibit cyclooxygenase enzymes in the brain from producing prostaglandins (Mirrasekhian et al., 2018). It has been postulated that disruption in the signaling of prostaglandin E2 pathway (due to genetic defects or exposure to environmental factors) during critical periods of brain development can lead to neurodevelopmental disorders such as ASD (Tamiji & Crawford, 2011). Mouse models have shown that abnormal prostaglandin ED signaling and cyclooxygenase 2 is related to ASD behavior and pathology (Wong et al., 2019; Rai-Bhogal et al., 2018). In the pre- and post-natal periods, alterations in lipid signaling pathways can negatively impact neurodevelopment and lead to autism. Studies collectively suggest that lipid signaling may play an important role in the pre- and postnatal periods, and alterations of this pathway can negatively impact the development of the nervous system and lead to ASD (Tamiji & Crawford, 2011).

D.5. Endocannabinoid Dysfunction

A proposed mechanism of APAP's analgesic action is modulation of the endocannabinoid system (Bauer et al., 2018). APAP can adversely affect fetal brain development by altering the cannabinoid system. (Angelis et al., 2021). APAP is thought to produce analgesic effects through the activation of CB_1 receptors, and in blocking these receptors, the analgesic effect of APAP is

removed. In the CNS, APAP metabolism produces para-aminophenol, which combines with arachidonic acid to produce *N*-arachidonoylphenolamine (AM404). This reaction is catalyzed by fatty acid amide hydrolase (FAAH), a central enzyme of the endocannabinoid system that catabolizes anandamide and 2-arachidonoylglycerol (both endocannabinoids). AM404 blocks the reuptake of anandamide, acting as an indirect agonist against the CB_1 receptors. Repeated exposure to APAP sets the level of anandamide lower, and lower levels are observed as decreased endocannabinoid tone in ASD and related disorders (Schultz et al., 2021). Thus, APAP may interfere with the endocannabinoid system during in utero development inducing long-lasting functional alterations in the developing brain.

The CB_1 receptors play an important role in neuron differentiation, axonal migration, and the establishment of neuronal connectivity (Bauer et al., 2018; Wilson & Nicoll, 2002; Doenni et al., 2016; Basavarajappa et al., 2009). Abnormal brain connectivity in children with ASD may be due to lack of CB1 axon guidance (Schultz & Gould, 2016; Schultz, 2010; McFadden and Minshew, 2013). The CB_2 receptors are important in immune system regulation and occur in immune and microglial cells, but are not affected significantly by APAP (Bauer, et al., 2018; Di Marzo, et al., 2004). APAP is metabolized in the liver to a number of different compounds. One of these compounds, AM404, is an agonist of cannabinoid receptors and may be responsible for the analgesic effects of APAP. AM404 and p-aminophenol, another APAP metabolite, have been shown to be toxic to mouse embryonic cortical neurons (Schultz et al., 2012). In another animal study, acute intraperitoneal administration of APAP showed changes in social behaviors in mice and corresponding elevations in cortical levels of endocannabinoids (Gould et al., 2012).

Of note, the endocannabinoid system is involved in the pathophysiology of ASD (Brigida et al., 2017; Schultz et al., 2021) and may also be involved in the development of ADHD and

related disorders of impulsivity due to its involvement in behavioral inhibition and reward-seeking behaviors (Lafenêtre et al., 2009; Navarrete et al., 2020; Schultz et al., 2019, 2021; Brunkhorst-Kannan et al., 2021; Klinger-Gratz et al., 2018). Thus, APAP may interfere with the endocannabinoid system during in utero development inducing long-lasting functional alterations in the developing brain.

Animal studies provide support for the biological plausibility of endocannabinoid disruption as a mechanism of action. In the BTBR autism mouse model, APAP acts like a cannabinoid and works to improve sociability by increasing the level of anandamide in the frontal cortex (Gould et al., 2012). Thus, damage caused by APAP in utero can be repaired by its use post-natally. This mechanism of a drug causing damage prenatally and then being useful as a treatment postnatally is not uncommon.

This is analogous to the valproic acid (VPA; Depakote) model of ASD. In this VPA animal model of ASD, rats treated with VPA have decreased sociability, which is linked to lower levels of anandamide in the brain (Nicolini & Fahnestock, 2018). Behavioral damage was observed in male rats compared to female rats, consistent with the male:female ratio observed in ASD. Treatments that inhibit the destruction of endocannabinoid anandamide can rescue these social deficits. Prenatal usage of valproate by mothers increases their risk of having children with ASD (Christensen et al., 2013, 2019), and conversely valproate rescues behavioral deficits in children with ASD (Hollander et al., 2001, 2005, 2010). APAP use in animal models causes toxicity and inhibition of fetal testosterone development that significantly disrupts fetal brain development. It also results in the inhibition of cyclooxygenase 2, which can affect long-term potentiation, spatial learning and cerebellar development.

D.6. Endocrine Disruption

The endocrine system consists of a network of hormone-producing glands and organs. The hormones produced in the endocrine system regulate multiple physiological processes, including growth, metabolism, and reproduction. Many of the hormones secreted, including thyroid hormone and growth hormone, cortisol, prolactin, and gonadotropins, are important for fetal brain development. However, endocrine-disrupting pharmaceuticals may interrupt hormone signaling that regulates fetal brain development (Frye et al., 2012). Notably, endocrine disrupters pose the greatest risk during prenatal development, and some may alter neural transmission and formation of neuronal networks (Kajta & Wojtowicz, 2013). Although the current evidence is still being developed, study of other environmental insults that cause fetal endocrine disruption resulting in ASD or ADHD symptoms is being completed, and researchers note that every effort should be made to reduce exposure to endocrine disrupting compounds (Long et al., 2019; Mari-Brauset et al., 2018).

D.7. Altered Brain-Derived Neurotropic Factor

Brain-derived neurotrophic factor (“BDNF”) is a protein that is widely expressed in the brain and is involved in several important neurodevelopmental processes and plays a role in the growth, survival, and maintenance of neurons and the nervous system (Barde, 1982). BDNF is released by neurons in response to activity and other stimuli (Huang, 2023). BDNF is implicated in synaptic plasticity, learning, memory, and attention (Garcia et al., 2012). Viberg et al. demonstrated that acute neonatal exposure to APAP during neonatal brain development in mice was shown to affect BDNF levels in the neonatal brain and indicate long-lasting impairment in cognitive function and analgesic and anxiolytic response in adult male mice (Viberg et al., 2014). These behavioral alterations in adulthood may be due to APAP-induced changes to BDNF levels

in the brain during a critical time in development. Studies have shown that dysregulation of BDNF is involved in ASD and ADHD (Bryn et al., 2015; Liu et al., 2015; Meng et al., 2017). BDNF alterations caused by APAP in utero may ultimately be responsible for behavioral and cognitive alterations during childhood that persist into adulthood.

D.8. Epigenetic Effects

Epigenetics is the study of heritable changes in gene expression (that is, turning genes "on" or "off") that are caused by mechanisms other than changes in the underlying DNA sequence (Bernstein et al., 2007). These changes can be caused by environmental factors, such as diet, stress, and exposure to toxins, and they can be passed from parent to offspring. DNA methylation is a type of epigenetic change that works by adding a chemical group to DNA, where it blocks certain proteins that attach to the DNA to "read" the gene, which effectively turns the gene "off." The chemical group can be removed from the DNA through demethylation. During brain development, epigenetic alterations like DNA methylation are important in regulating gene expression (Jeong et al., 2021; Spiers et al., 2015). Brain development involves dynamic orchestration of gene expression, and neurogenesis, neuronal differentiation, and synaptic plasticity are all epigenetically regulated (Spiers et al., 2015).

Defects in certain epigenetic marks can result in neurodevelopmental disorders and affect brain development (Jakovcevski, 2012). Modifications to the epigenome can result in abnormal gene expression, which may lead to neurodevelopmental disorders like ASD and ADHD (Reichard & Zimmer-Bensch, 2021). Carter and Blizard examined whether certain autism genes are targeted by environmental factors and analyzed gene/environment interactions in 206 autism-susceptible genes from the Autworks database to interrogate over one million gene/chemical interactions in the comparative toxicogenomics database (Carter & Blizard, 2016). APAP was identified as a drug

implicated in autism and showed a significant degree of bias towards ASGs. Overall, APAP modified gene expression in 92 ASGs and was a drug with one of the most significant enrichment scores, second-highest after Depakote (valproate), which has a "black box warning"[2] for the fetal risk of neural tube defects (Carter & Blizard, 2016).

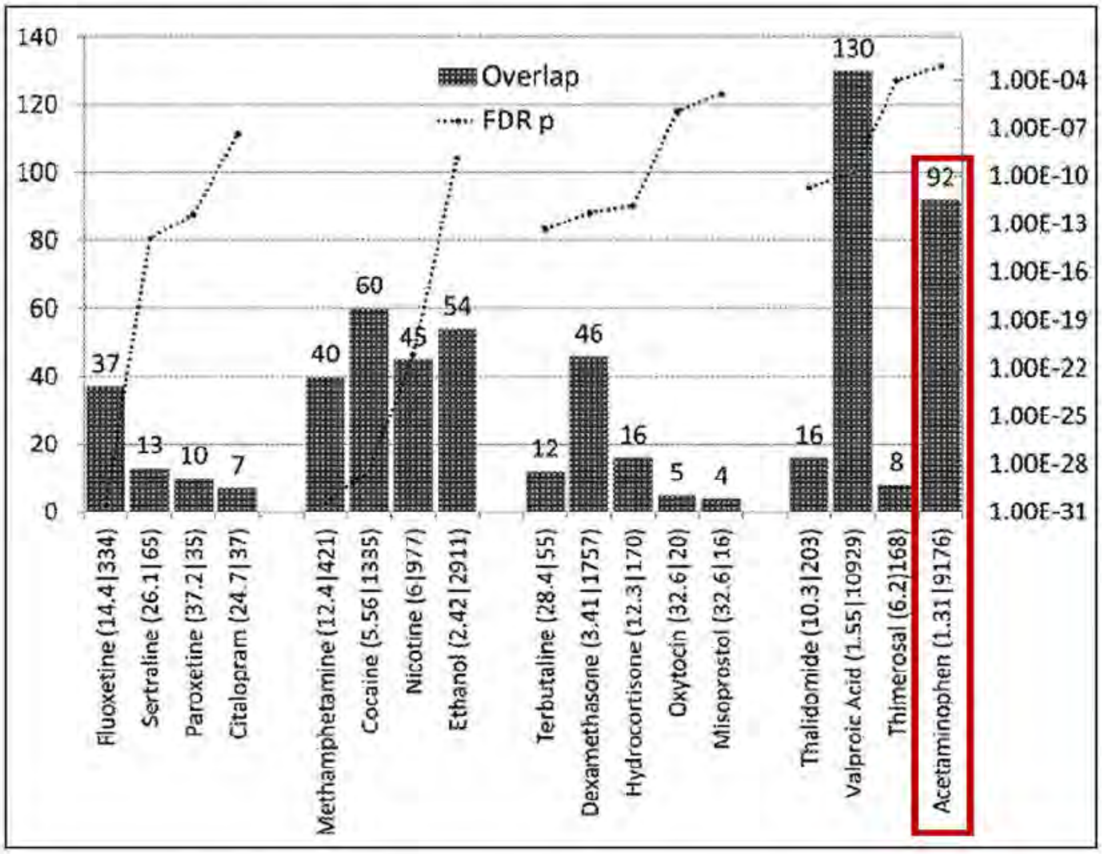


Fig. 2. The number of ASGs affected by drugs implicated in autism (left axis), and p values (right axis). The enrichment ratio and the total number of genes affected by each compound are shown

[2] Black box warnings (BBWs) are the most potent safety advisories regarding medications that are included in a drug's labeling information as mandated by the Food and Drug Administration (FDA) (Panagiotou et al., 2016). These warnings draw attention to significant risks associated with the use of the drug. The FDA determines whether a black box warning is necessary based on evidence from animal studies, clinical trials, and post-marketing surveillance. From 2005 to 2008, approximately 14% of safety labeling changes involved the addition or modification of black box warnings. Within 25 years of approval, around 20% of approved drugs either receive a black box warning or are withdrawn from the market due to serious adverse effects. Currently, there are over 400 drugs that carry black box warnings, indicating an almost twofold increase in the past 15 years.

after each compound name. First batch = SSRI antidepressants, second = drugs of abuse, third = drugs used during labor, fourth = others (Carter & Blizard, 2016).

Use of APAP during pregnancy has been associated with epigenomic alterations, specifically DNA methylation changes in the placenta, newborn blood, and fetal tissues (Eslamimehr et al., 2022; Addo et al., 2019). In an epigenome-wide association study, Gervin et al. examined whether long-term prenatal exposure to APAP was associated with DNA methylation changes, an epigenomic alteration, changes in cord blood, and clinical ADHD diagnoses in samples selected from the Norwegian Mother and Child Cohort (MoBa) (Gervin et al., 2017). The study found increased differential DNA methylation in genes involved in oxidative stress and neurotransmission (but not ADHD specifically) in children diagnosed with ADHD. Gervin et al. concluded that the study results were compatible with epidemiological evidence showing an increased risk of developing ADHD with long-term fetal exposure to APAP. This is consistent with the finding that APAP modified gene expression in 92 ASGs.

Spildrejorde et al. investigated APAP's effects on a model of early human brain development using a multi-omics approach, which combined datasets from multiple disciplines to gain a more comprehensive understanding of the developing brain (Spildrejorde et al., 2022). Human embryonic stem cells undergoing in vitro neuronal differentiation were exposed to daily media changes with APAP amounts comparable to maternal therapeutic doses. This resulted in APAP-induced chromatin-opening changes linked to gene expression. These methylated and/or expressed genes were related to signaling, neurotransmission, and cell fate-determination. Spildrejorde et al. concluded that, based on the data, APAP may play a causal role in impaired neurodevelopment. Like the other studies reviewed here, epigenome alterations occurring during fetal development can affect neurodevelopment and lead to neurodevelopmental diseases. As these studies show, fetal exposure to APAP can lead to epigenetic changes in a large number of autism-

susceptible genes (earning it the second highest enrichment score), which constitutes a mechanism of action for neurodevelopmental disorders like ADHD and ASD.

V. OPINIONS

Based on my expertise, experience, medical training, and review of the discussed scientific materials, I state the following opinions:

1. As recognized by the transdiagnostic approaches to neurodevelopmental disorders, there is a strong interrelationship between the neurodevelopmental disorders of ASD and ADHD and across neurodevelopmental disorders.
2. Based on this interconnectedness of neurodevelopmental disorders, including ADHD and ASD, it is appropriate to review the body of evidence that measures neurodevelopmental disorders and to not limit the analysis to studies that focus on ASD and ADHD as specified outcomes.
3. I reviewed the underlying epidemiological literature regarding whether prenatal use of APAP can cause the neurodevelopmental disorders of ASD and ADHD, and that body of literature is consistent with my opinion that to fully investigate and address the question of whether prenatal use of APAP can cause the neurodevelopmental disorders of ASD and ADHD, scientists should look to the full body of neurodevelopmental studies.
4. There are multiple, plausible mechanisms of action to explain how APAP can impact fetal brain development and lead to neurodevelopmental disorders in offspring.
5. Depending on the timing and duration of APAP exposure to the developing brain, if the suspect mechanisms of injury occur, then one would expect a wide variety of diffuse neurologic symptoms/injuries when the brain develops.
6. Based on my review of the literature, the expert reports, and my decades of clinical experience, I would not advise my pregnant patients to take APAP unless they have fever

and to use at the lowest effective dose for the shortest time possible and the lowest possible frequency.

VI. CONCLUSION

In conclusion, as a psychiatrist with a focus in neuropsychopharmacology, it is my opinion that it is appropriate to review the comprehensive evidence that assesses the causal association between exposure to toxic substances and neurodevelopmental disorders when assessing a casual association between an exposure and the specific neurodevelopmental disorders of ASD and ADHD. That is true to assess the causal association between prenatal exposure to APAP and the neurodevelopmental disorders of ASD and ADHD. In addition, I find there are number of plausible biological mechanisms to explain the causal association between prenatal exposure to APAP and the neurodevelopmental disorders of ASD and ADHD. Finally, based on my review of the literature, and the expert reports, as well as decades of clinical experience, I would not advise my pregnant patients to take APAP unless they have fever and to use at the lowest effective dose for the shortest time possible and the lowest possible frequency. I hold these opinions to a reasonable degree of medical and scientific certainty.

Appendix 1

DSM-5, DSM-IV, and ICD for ASD

DSM-5 Criteria for ASD:

(A) Persistent deficits in social communication and social interaction across multiple contexts, as manifested by the following, currently or by history:

   i. Deficits in social-emotional reciprocity, ranging for example from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions.
   ii. Deficits in nonverbal communicative behaviors used for social interaction, ranging, for example, from poorly integrated verbal and nonverbal communication to abnormalities in eye contact and body language or deficits in understanding and use of gestures; to a total lack of facial expressions and nonverbal communication.
   iii. Deficits in developing, maintaining, and understanding relationships, ranging, for example, from difficulties adjusting behavior to suit various social contexts; to difficulties in sharing imaginative play or in making friends; to absence of interest in peers.

(B) Restricted, repetitive patterns of behavior, interests, or activities, as manifested by at least two of the following, currently or by history:

   i. Stereotyped or repetitive motor movements, use of objects, or speech (e.g., simple motor stereotypies, lining up toys or flipping objects, echolalia, idiosyncratic phrases).
   ii. Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behaviors (e.g., extreme distress at small changes, difficulties with transitions, rigid thinking patterns, greeting rituals, need to take same route or eat same food every day).
   iii. Highly restricted, fixated interests that are abnormal in intensity or focus (e.g., strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interests).
   iv. Hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment (e.g., apparent indifference to pain/temperature, adverse response to specific sounds or textures, excessive smelling or touching of objects, visual fascination with lights or movement).

(C) Symptoms must be present in the early developmental period (but may not become fully manifest until social demands exceed limited capacities, or may be masked by learned strategies later in life).

(D) Symptoms cause clinically significant impairment in social, occupational or other important areas of current functioning.

(E) These disturbances are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay.

The DSM-5 diagnostic criteria also allow for the following specifiers: comorbid intellectual impairment, language impairment; known medical or genetic condition or environmental factors; associated with another neurodevelopmental, mental, or behavioral disorder; or with catatonia.

DSM-IV-TR Criteria for ASD:

1. Autistic Disorder:
   a. A total of six (or more) items from (1), (2) and (3), with at least two from (1) and one each from (2) and (3):
      i. Qualitative impairment in social interaction, as manifested by at least two of the following:
         1. Marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
         2. Failure to develop peer relationships appropriate to developmental level
         3. A lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest)
         4. Lack of social or emotional reciprocity
      ii. Qualitative impairments in communication as manifested by at least one of the following:
         1. Delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime)
         2. In individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others
         3. Stereotyped and repetitive use of language or idiosyncratic language
         4. Lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level
      iii. Restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:
         1. Encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
         2. Apparently inflexible adherence to specific, nonfunctional routines or rituals
         3. Stereotyped and repetitive motor manners (e.g., hand or finger flapping or twisting, or complex whole-body movements)
         4. Persistent preoccupation with parts of objects
   b. Delays or abnormal functioning in at least one of the following areas, with onset prior to age 3 years: (1) social interaction, (2) language as used in social communication, or (3) symbolic or imaginative play
   c. The disturbance is not better accounted for by Rett Disorder or Childhood Disintegrative Disorder

2. Asperger's Disorder:
   a. Qualitative impairment in social interaction, as manifested by at least two of the following:

i. Marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulation social interaction
ii. Failure to develop peer relationships appropriate to developmental level
iii. A lack of spontaneous seeking to share enjoyment, interests, or achievement with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest to other people)
iv. Lack of social or emotional reciprocity

b. Restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:
i. Encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
ii. Apparently inflexible adherence to specific, nonfunctional routines or rituals
iii. Stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements)
iv. Persistent preoccupation with parts of objects

c. The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning
d. There is no clinically significant general delay in language (e.g., single words used by age 2 years, communicative phrases used by age 3 years)
e. There is no clinically significant delay in cognitive development or in the development of age-appropriate self-help skills, adaptive behavior (other than in social interaction), and curiosity about the environment in childhood.
f. Criteria are not met for another specific Pervasive Developmental Disorder or Schizophrenia

3. Pervasive Developmental Disorder – Not Otherwise Specified (including atypical autism):
a. This category should be used when there is a severe and pervasive impairment in the development of reciprocal social interaction associated with impairment in either verbal or nonverbal communication skills or with the presence of stereotyped behavior, interests, and activities, but the criteria are not met for a specific Pervasive Developmental Disorder, Schizophrenia, Schizotypal Personality Disorder, or Avoidant Personality Disorder. For example, this category includes “atypical autism” – presentations that do not meet the criteria for Autistic Disorder because of late age onset, atypical symptomatology, or subthreshold symptomatology or all of these.

ICD Classifications for ASD:

1. ICD-9: The ICD-9 is a previous version of the global standard for classifying diseases and medical conditions (WHO, *International Classification of Diseases, 9th Revision*, 1977) (ICD-9)). It was widely used by healthcare professionals and organizations until 2015, when it was replaced by the ICD-10 in many countries. In the ICD-9 coding system, autism was classified as “299.0 - Infantile Autism.” The diagnostic criteria were not explicitly provided within the coding system itself, since the ICD-9 system primarily focused on classifying and organizing diseases and disorders.

2. ICD-10: Autism is classified under ICD-10 Chapter V, “Mental and Behavioural Disorders, Disorders of Psychological Development (F8089)” (WHO, *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines*, 1992 (ICD-10)). The disorders in Chapter V are described as having the following in common: “(a) onset invariably during infancy or childhood, (b) impairment or delay in development of functions that are strongly related to biological maturation of the central nervous system, and (c) a steady course without remission or relapses” (ICD-10). Autism is listed under the Pervasive Developmental Disorders (“PDD”) subcategory, code F84, which includes eight different disorders.[3] Notably, these PDDs are defined similarly as they were in the DSM-IV-TR, as “characterized by qualitative abnormalities in reciprocal social interactions and in patterns of communication, and by a restricted, stereotyped, repetitive repertoire of interests and activities. These qualitative abnormalities are a pervasive feature of the individual’s functioning in all situations” (ICD-10). This system of discrete diagnoses is comparable to that in the DSM-IV-TR. However, the single Autism Spectrum Disorder category in the DSM-5 significantly contrasts the eight Pervasive Developmental Disorders in ICD-10.

3. ICD-11: The ICD-11 was adopted in 2019 but has not yet been implemented in the United States (WHO, *International Statistical Classification of Diseases and Related Health Problems*, 11th Revision, 2018 (ICD-11)). It includes updated criteria for autism spectrum disorders. Importantly, the ICD-11 criteria for autism differ slightly from the criteria used in other diagnostic systems such as the DSM-5. The diagnostic criteria for autism, Code 6A02–Autism Spectrum Disorder, in the ICD-11 are as follows:
    i. Social Communication and Social Interaction Difficulties: Difficulty with social-emotional reciprocity, such as abnormal social approach, reduced sharing of interests or emotions, and difficulty with back-and-forth conversation.
    ii. Deficits in nonverbal communicative behaviors, such as abnormal eye contact, body language, and gestures.

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[3] The eight different disorders are: Childhood Autism (F84.0); Atypical Autism (F84.1); Rett Syndrome (F84.2); Other Childhood Disintegrative Disorder (F84.3); Overactive Disorder Associated with Mental Retardation and Stereotyped Movements (F84.4); Asperger Syndrome (F84.5); Other Pervasive Developmental Disorders (F84.8); and Pervasive Developmental Disorder, Unspecified (F84.9).

   iii. Challenges in developing and maintaining relationships, including difficulty adjusting behavior to suit different social contexts and problems with imaginative play or making friends.
b. Restricted and Repetitive Patterns of Behavior, Interests, or Activities:
   i. Stereotyped or repetitive motor movements, use of objects, or speech.
   ii. Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior.
   iii. Highly restricted, fixated interests that are abnormal in intensity or focus.
   iv. Hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of the environment.
c. Symptoms must be present in early childhood but may not become fully manifest until social demands exceed limited capacities.
d. Symptoms cause significant impairment in personal, social, educational, occupational, or other important areas of functioning.
e. These difficulties are not better explained by intellectual disability or global developmental delay.

ICD-11 also discusses environmental, genetic and physiological risk and prognostic factors, in addition to culture- and gender-related diagnostic issues.

Appendix 2

DSM-5, DSM-IV, and ICD for ADHD

DSM-5 Criteria for ADHD:

A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by inattention and/or hyperactivity and impulsivity.

1. Inattention: Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities. Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.
    a. Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
    b. Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).
    c. Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction).
    d. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
    e. Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines).
    f. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).
    g. Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys paperwork, eyeglasses, mobile telephones).
    h. Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).
    i. Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

2. Hyperactivity and impulsivity: Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities. Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or a failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.
    a. Often fidgets with or taps hands or feet or squirms in seat.

b. Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office, or other workplace, or in other situations that require remaining in place).
c. Often runs about or climbs in situations where it is inappropriate, (Note: in adolescents or adults, may be limited to feeling restless).
d. Often unable to play or engage in leisure activities quietly.
e. Is often "on the go," acting as if "driven by a motor" (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).
f. Often talks excessively.
g. Often blurts out an answer before a question has been completed (e.g., completes people's sentences, cannot wait for turn in conversation).
h. Often has difficulty waiting his or her turn (e.g., while waiting in line).
i. Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing).

In addition, the following conditions must be met under the DSM-5:

i. Several inattentive or hyperactive-impulsive symptoms were present prior to age 12 years.
ii. Several inattentive or hyperactive-impulsive symptoms are present in two or more settings (e.g., at home, school, or work; with friends or relatives; in other activities).
iii. There is clear evidence that the symptoms interfere with, or reduce the quality of, social, academic, or occupational functioning.
iv. The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better explained by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, personality disorder, substance intoxication or withdrawal).

Based on the type of symptoms that are exhibited by the child, there are three different presentations of ADHD, which the clinician should specify:

1. <u>Combined presentation</u>: If both inattention and hyperactivity-impulsivity are met for the past 6 months.
2. <u>Predominantly inattentive presentation</u>: If inattention is met but hyperactivity-impulsivity is not met for the past 6 months.
3. <u>Predominantly hyperactive/impulsive presentation</u>: If hyperactivity-impulsivity is met, but inattention is not met for the past 6 months.

The clinician should also specify the severity of the symptoms and if the child is in partial remission.

ICD Classifications for HKD:

1. ICD-9: In the ICD-9, ADHD falls under the broader category of "Hyperkinetic Disorders." The specific ICD-9 codes related to ADHD are as follows: 314.0: Attention Deficit Disorder without mention of hyperactivity (predominantly inattentive type); and 314.1: Attention Deficit Disorder with hyperactivity (combined type, overactivity NOS, predominantly hyperactive/impulsive type, or simple disturbance of attention with overactivity).

2. ICD-10: In the ICD-10, ADHD is categorized under the section "Hyperkinetic Disorders (F90)." The ICD-10 code for ADHD is F90.0, known as "Disturbance of Activity and Attention." The term "attention deficit" encompasses various definitions such as attention deficit disorder with hyperactivity, attention deficit hyperactivity disorder, or attention deficit syndrome with hyperactivity (Doernberg & Hollander, 2016). Significantly, the ICD-10 excludes the diagnosis of ADHD when it coexists with HKD associated with conduct disorder (F90.1). Furthermore, pervasive developmental disorders are classified as exclusionary for HKD in the ICD-10. This contrasts with the DSM-5, which allows for a comorbid diagnosis of ADHD and autism.

3. ICD-11: As previously mentioned, ICD-11 has not yet been implemented in the United States despite its adoption in 2019. In ICD-11, hyperkinetic disorder was removed and renamed attention-deficit hyperactivity disorder (ADHD). ADHD is classified under the category of Neurodevelopmental Disorders and was given code 6A05-Attention Deficit Hyperactivity Disorder.

Appendix 3

Assessments for ASD and ADHD

The following assessments are used to assess for ASD and ADHD symptoms in children:

A. Developmental and Well-Being Assessment (DAWBA): A novel package of questionnaires, interviews, and rating techniques designed to examine ICD-10, DSM-IV and DSM-5 diagnoses in individuals between the ages of 2 and 65 years old. Information is collected from up to three sources (1) interview with the parents of 2 to 17 year olds; (2) interview with 11to 17 year olds themselves; (3) questionnaires completed by teachers of 2 to 17 year olds (*DAWBA | Youthinmind*, n.d.; Goodman et al., 2000). Interviews can be completed via computer or in interview format. Common emotional, behavioral, and hyperactivity disorders in addition to severe disorders are examined. Validation studies have shown the DAWBA to be a good measure for both epidemiological studies and in clinic assessments. DAWBA has been shown to be an accurate and sufficient measure to assess for ADHD in clinical community settings, without direct patient contact by the diagnosing clinician (Foreman et al., 2009), and has good agreement with the Autism Diagnostic Interview – Revised (ADI-R), a gold standard diagnostic caregiver interview used to diagnose ASD (Murphy et al., 2018). Most importantly, this questionnaire can be completed without direct patient contact, making it ideal for community settings and large-scale epidemiological studies.

B. Child Behavior Checklist (CBCL): The CBCL is a component of the Achenbach System of Empirically Based Assessment (ASEBA), which is used to detect emotional, social and behavioral problems in children and adolescents (Mazefsky et al., 2011; *School-Age (CBCL, TRF, YSR)*, n.d.). There are multiple versions of this questionnaire, including (1) parent/caregiver report form, (2) teacher's report form, and (3) youth self-report, which can be completed for different age groups including (1) ages 1 ½ to 5 years (parent/caregiver and teacher form), (2) ages 6 to 18 years (parent/caregiver and teacher form), (3) ages 11 to 18 years (youth self-report form). All forms were revised from their 1991 versions in 2000 or 2001. The preschool CBCL 1 ½ to 5 years forms include syndrome scales (emotionally reactive; anxious/depressed; somatic complaints; withdrawn; sleep problems; attention problems; aggressive behavior) and DSM-oriented scales (depressive problems, anxiety problems, autism spectrum problems; attention deficit/hyperactivity problems; oppositional defiant problems). The school-age CBCL 6 to 18 years and youth self-report 11 to 18 years include the following syndrome scales: anxious/depressed, withdrawn/depressed, somatic complaints, social problems, thought problems, attention problems, rule breaking behavior and aggressive behavior; and DSM-oriented scales: depressive problems, anxiety problems, somatic problems, attention deficit/hyperactivity problems, oppositional defiant problems, conduct problems. The CBCL 1 ½ to 5 years parent/caregiver and teacher forms DSM-ASD (previously DSM-pervasive developmental problems, DSM-PDD) subscale has been validated as a good Level 1 screener of ASD with good discriminatory questions and measurement invariance across multiple diverse populations (International ASEBA Consortium et al., 2020). Although ASD is not included in the syndrome scales for school-age children, the CBCL 6 to 18 years parent/caregiver and teacher forms have also been validated as appropriate screeners for ASD when

examining scores on specific subscales, including the Thought and Social Problems scales (Mazefsky et al., 2011), and the Withdrawn/Depressed, Social Problems and Attention Problems scales (Arias et al., 2022). The CBCL DSM-oriented subscale for ADHD and the Attention Problems subscale have both been validated as diagnostic measures and screeners for ADHD, with good predictability for later diagnoses of the disorder (Biederman et al., 2021; Oerbeck et al., 2020; Spencer et al., 2018).

C. Childhood Autism Spectrum Test (CAST; formerly known as the Childhood Asperger Screening Test): CAST is a 37-item parent/caregiver questionnaire developed in 2002 as a screening measure for autism spectrum symptoms. The CAST measures difficulties and preferences in social and communication skills, including social interaction, reciprocity, eye contact, play activities, and restricted and repetitive behaviors. Validity and test-retest reliability has been examined in multiple large population-based observational studies that include samples from general populations of school-aged children. It shows good sensitivity and specificity for autism spectrum symptoms with a moderate positive predictive value. A cut-off point of 15 has been used; at this level there is a sensitivity of 100%, specificity of 50%, and positive predictive value of 50% (*The Childhood Asperger Syndrome Test (CAST)*, n.d.; J. Williams et al., 2005, 2006; J. G. Williams et al., 2008).

D. Conners Parent Rating Scale- Revised short form (CPRS-R:S): The CPRS-R:S is a parent/caregiver report questionnaire to measure behavioral challenges in children and adolescents aged 3 to 17. The Conners rating scales include long and short forms that can be administered to children and adolescents. The short form has 27 items that parents/caregivers will rate on a Likert scale to indicate how often their child engages in the behaviors listed (never to very often). The Conners rating scales are useful for the assessment of children and adolescents with ADHD, as it assesses the 12 criteria listed in the DSM-IV for ADHD (Kumar & Steer, 2003). The ratings are summed to yield a 6-item oppositional, 6-item cognitive problems/inattention and 6-item hyperactivity scale. It also includes the ADHD Index, which includes items found to best discriminate youth with ADHD from neurotypical peers. The Conners rating scale system was revised as the Conners-3 in 2008 (*Conners 3rd Edition*, n.d.).

E. Conners Kiddie Continuous Performance Test (Conners K-CPT): The Conners K-CPT is a computerized assessment that evaluates attention, function, reaction time, accuracy and impulse control. The original K-CPT was developed to evaluate ADHD symptoms in children aged 4 to 5 years. The K-CPT 2 was first developed in 2001, and is currently used for children ages 4 to 7:11 years old. There is also a version used in children 8 years and older, called the Conners Continuous Performance Test 3rd Edition (CPT-3), which was developed in 2014. Measures on the K-CPT include commission errors, omission errors, hit reaction time standard error and detectability. This measure is frequently used in epidemiological and clinical research, and the variables have been correlated to ADHD symptoms (Epstein et al., 2003; Conners et al., 2003; Breaux et al., 2016).

F. Strengths and Difficulties Questionnaire (SDQ): The SDQ is a 25-item questionnaire measuring emotional and behavioral difficulties in children and adolescents aged 4 to 17 years from the perspective of the parent/caregiver, teacher, or child/adolescent (aged 11 to

17). It can be used as both a screening or diagnostic measure, and as an outcome measure in treatment trials. The SDQ is widely used across the globe in both clinical and research settings, has been translated into 40 different languages, and has been well-validated in multiple populations. It has five scales scored from 0 to 10; emotional problems, conduct problems, hyperactivity, peer problems and prosocial behaviors. The scales are combined (minus the prosocial scale) into a "total difficulties" score (0 to 40). The SDQ is shown to be a valid tool for discriminating cases with ADHD from those without ADHD or with other mental health diagnoses in large population-based studies, including those that had confirmed diagnoses via a medical/healthcare professional (Algorta et al., 2016; Russell et al., 2013). It has also been studied in ASD populations and in individuals with co-morbid ASD and ADHD, with good sensitivity and specificity reported.

G. <u>The DSM-ADHD Questionnaire:</u> Symptom checklists have been developed for ADHD and other disorders based on their diagnostic criteria in the DSM (DSM-IV, DSM-IV-TR, DSM-5). The DSM nosology has been validated as a way to differentiate symptoms and diagnose preschoolers, in addition to older children and adults (Sterba et al., 2007).

The foregoing opinions are substantively identical to the opinions stated in my June 16, 2023 report. All opinions offered herein are held to a reasonable degree of scientific certainty.

Dated: June 22, 2023

Respectfully submitted,

Eric Hollander MD

Eric Hollander, M.D.

RULE 26 GENERAL CAUSATION EXPERT REPORT OF DR. ERIC HOLLANDER, M.D., DFAPA, FACNP

Materials Considered

Expert Reports:

Expert Report of Dr. Andrea Baccarelli, M.D., PhD
Expert Report Dr. Robert Cabrera, PhD
Expert Report Dr. Stan Louie, PharmD
Expert Report Dr. Brandon Pearson, MSc, PhD

Depositions:

Deposition of Carol Jeffcoat (5/25/2023) and Attached Exhibits
Deposition of Edwin Kuffner (5/24/2023) and Attached Exhibits
Deposition of Islah Ahmed (6/1/2023) and Attached Exhibits
Deposition of Leslie Shur (5/26/2023) and Attached Exhibits
Deposition of Rachel Weinstein (5/19/2023) and Attached Exhibits

Drug Labels:

Depakote Label (2023)
Paracetamol 500mg Tablets (POM) Label
Ultracet Label
Tylenol Acetaminophen Extra Strength Label
Tylenol Regular Strength Label
TYLENOL® Professional. "Safety Profile of TYLENOL®." Accessed June 16, 2023. https://www.tylenolprofessional.com/safety-and-efficacy/safety.
TYLENOL® Professional. "TYLENOL® Dosing for Infants, Children & Adults." Accessed March 23, 2023. https://www.tylenolprofessional.com/adult-dosage.

FDA Documents:

All FDA Productions in This Case
FDA, Acetaminophen: Background and Overview (6/29/2009)
FDA, Drug Therapeutics & Regulation in the U.S. (1/31/2023),
https://www.fda.gov/about-fda/fda-history-exhibits/drug-therapeutics-regulation-us
Internal Analgesic, Antipyretic, and Antirheumatic Drug Products for Over-the-Counter Human Use; Tentative Final Monograph, 53 FR 46204-01, 1988 WL 275236 (F.R.)
Ofirmev FDA Drug Approval Package, https://www.accessdata.fda.gov/drugsatfda_docs/nda/2010/022450_ofirmev_toc.cfm.

JJCI-Produced Documents:

Company Core Data Sheets, including:
Company Core Data Sheet (APAP-JJCI-0000269165-95)
Company Core Date Sheet Version 2.0 (APAP-JJCI-0000749925-974)
Company Core Date Sheet Version 3.0 (APAP-JJCI-0000269231-250)
Company Core Date Sheet Version 4.0 (APAP-JJCI-0000269251-271)
Company Core Date Sheet Version 5.0 (APAP-JJCI-0000269272-300)
Company Core Date Sheet Version 6.0 (APAP-JJCI-0000241259-287)
Company Core Date Sheet Version 7.0 (APAP-JJCI-0000208431-464)

Periodic Benefit Risk Evaluation Report/Periodic Safety Update Report (APAP-JJCI 0000753540)
Project Cocoon Forest Plots, Redrawn
Project Cocoon Nonsystematic Reviews
Project Cocoon Update, August 2022
Project Cocoon Repro Slides
Suarez, et al. Article (APAP-JJCI-0000206374-85)

Laws, Regulations, and Rules:

21 C.F.R. 201
Federal Register, Vol. 73, No. 104 (May 29, 2008)
Federal Food, Drug, and Cosmetic Act Amendments (Durham-Humphrey amendments), Pub. L. No. 82-215, 65 Stat. 648 (1951).

Scientific Articles & Other Publications:

"A 14-Month Randomized Clinical Trial of Treatment Strategies for Attention-Deficit/Hyperactivity Disorder." *ARCH GEN PSYCHIATRY* 56 (1999).

"A Review of the Pathophysiology, Etiology, and Treatment of Attention-Deficit Hyperactivity Disorder (ADHD)." Accessed May 17, 2023. https://doi.org/10.1177/1060028013510699.

Abdallah, M. W., E. L. Mortensen, K. Greaves-Lord, N. Larsen, E. C. Bonefeld-Jørgensen, B. Nørgaard-Pedersen, D. M. Hougaard, and J. Grove. "Neonatal Levels of Neurotrophic Factors and Risk of Autism Spectrum Disorders." *Acta Psychiatrica Scandinavica* 128, no. 1 (2013): 61–69. https://doi.org/10.1111/acps.12020.

Abdallah, Morsi W., Nanna Larsen, Jakob Grove, Eva C. Bonefeld-Jørgensen, Bent Nørgaard-Pedersen, David M. Hougaard, and Erik L. Mortensen. "Neonatal Chemokine Levels and Risk of Autism Spectrum Disorders: Findings from a Danish Historic Birth Cohort Follow-up Study." *Cytokine* 61, no. 2 (February 2013): 370–76. https://doi.org/10.1016/j.cyto.2012.11.015.

Abdallah, Morsi W., Nanna Larsen, Jakob Grove, Bent Nørgaard-Pedersen, Poul Thorsen, Erik L. Mortensen, and David M. Hougaard. "Amniotic Fluid Inflammatory Cytokines: Potential Markers of Immunologic Dysfunction in Autism Spectrum Disorders." *The World Journal of Biological Psychiatry* 14, no. 7 (September 2013): 528–38. https://doi.org/10.3109/15622975.2011.639803.

Abdallah, Morsi W., Nanna Larsen, Erik L. Mortensen, Hjördis Ó. Atladóttir, Bent Nørgaard-Pedersen, Eva Cecilie Bonefeld-Jørgensen, Jakob Grove, and David M. Hougaard. "Neonatal Levels of Cytokines and Risk of Autism Spectrum Disorders: An Exploratory Register-Based Historic Birth Cohort Study Utilizing the Danish Newborn Screening Biobank." *Journal of Neuroimmunology* 252, no. 1–2 (November 2012): 75–82. https://doi.org/10.1016/j.jneuroim.2012.07.013.

ACOG. "Reducing Prenatal Exposure to Toxic Environmental Agents," July 2021.

"ACOG Response to Consensus Statement on Paracetamol Use During Pregnancy." Accessed June 13, 2023. https://www.acog.org/en/news/news-articles/2021/09/response-to-consensus-statement-on-paracetamol-use-during-pregnancy.

Addo, Kezia A, Catherine Bulka, Radhika Dhingra, Hudson P Santos, Lisa Smeester, T Michael O'Shea, and Rebecca C Fry. "Acetaminophen Use during Pregnancy and DNA Methylation in the Placenta of the Extremely Low Gestational Age Newborn (ELGAN) Cohort." *Environmental Epigenetics* 5, no. 2 (August 6, 2019): dvz010. https://doi.org/10.1093/eep/dvz010.

Ahmadalipour, Ali, Leila Mehdizadeh Fanid, Narges Zeinalzadeh, Maedeh Alizadeh, Helaleh Vaezi, Zahra Hassanpour Aydinlou, and Seyed Gholamreza Noorazar. "The First Evidence of an Association between a Polymorphism in the Endocannabinoid-Degrading Enzyme FAAH (FAAH Rs2295633) with Attention Deficit Hyperactivity Disorder." *Genomics* 112, no. 2 (March 2020): 1330–34. https://doi.org/10.1016/j.ygeno.2019.07.024.

Akintunde, Marjannie Eloi, Melissa Rose, Paula Krakowiak, Luke Heuer, Paul Ashwood, Robin H. Hansen, Irva Hertz-Picciotto, and Judy Van de Water. "Increased Production of IL-17 in Children with Autism Spectrum Disorders and Co-Morbid Asthma." *Journal of Neuroimmunology* 286 (September 15, 2015): 33–41. https://doi.org/10.1016/j.jneuroim.2015.07.003.

AL-Ayadhi, Laila Yousef, and Gehan Ahmed Mostafa. "Elevated Serum Levels of Interleukin-17A in Children with Autism." *Journal of Neuroinflammation* 9, no. 1 (December 2012): 595. https://doi.org/10.1186/1742-2094-9-158.

Aldinger, Kimberly A., Christianne J. Lane, Jeremy Veenstra-VanderWeele, and Pat Levitt. "Patterns of Risk for Multiple Co-Occurring Medical Conditions Replicate Across Distinct Cohorts of Children with Autism Spectrum Disorder." *Autism Research* 8, no. 6 (December 2015): 771–81. https://doi.org/10.1002/aur.1492.

Alemany, Silvia. "Prenatal and Postnatal Exposure to Acetaminophen in Relation to Autism Spectrum and Attention-Deficit and Hyperactivity Symptoms in Childhood: Meta-Analysis in Six European Population-Based Cohorts." *European Journal of*

Epidemiology 36 (May 2021): 12. https://doi.org/10.1007/s10654-021-00754-4.

Algorta, Guillermo Perez, Alyson Lamont Dodd, Argyris Stringaris, and Eric A. Youngstrom. "Diagnostic Efficiency of the SDQ for Parents to Identify ADHD in the UK: A ROC Analysis." *European Child & Adolescent Psychiatry* 25 (2016): 949–57. https://doi.org/10.1007/s00787-015-0815-0.

Ali, Shirin, Karen Klahr Miller, and Oliver Freudenreich. "Management of Psychosis Associated With a Prolactinoma: Case Report and Review of the Literature." *Psychosomatics* 51, no. 5 (September 2010): 370–76. https://doi.org/10.1016/S0033-3182(10)70718-0.

Allen, Andrea, Audrey King, and Eric Hollander. "Obsessive-Compulsive Spectrum Disorders." *Clinical Research* 5, no. 3 (2003).

Allison, Carrie, Jo Williams, Fiona Scott, Carol Stott, Patrick Bolton, Simon Baron-Cohen, and Carol Brayne. "The Childhood Asperger Syndrome Test (CAST): Test–Retest Reliability in a High Scoring Sample." *Autism* 11, no. 2 (March 2007): 173–85. https://doi.org/10.1177/1362361307075710.

Alwan, Sura, Elizabeth A. Conover, Lorrie Harris-Sagaribay, Steven H. Lamm, Sharon V. Lavigne, Shari I. Lusskin, Sarah G. Obican, Alfred N. Romeo, Anthony R. Scialli, and Katherine L. Wisner. "Paracetamol Use in Pregnancy—Caution over Causal Inference from Available Data." *Nature Reviews Endocrinology* 18, no. 3 (March 2022): 190–190. https://doi.org/10.1038/s41574-021-00606-x.

Amada, Naoki, Yuki Yamasaki, Claire M. Williams, and Benjamin J. Whalley. "Cannabidivarin (CBDV) Suppresses Pentylenetetrazole (PTZ)-Induced Increases in Epilepsy-Related Gene Expression." *PeerJ* 1 (November 21, 2013): e214. https://doi.org/10.7717/peerj.214.

Aman, Michael. "The Aberrant Behavior Checklist: A Behavior Rating Scale for the Assessment of Treatment Effects." American Journal of Mental Deficiency, 1985.

Aman, Michael G., Cristan A. Farmer, Jill Hollway, and L.Eugene Arnold. "Treatment of Inattention, Overactivity, and Impulsiveness in Autism Spectrum Disorders." *Child and Adolescent Psychiatric Clinics of North America* 17, no. 4 (October 2008): 713–38. https://doi.org/10.1016/j.chc.2008.06.009.

Ameis, Stephanie H., Jin Fan, Conrad Rockel, Aristotle N. Voineskos, Nancy J. Lobaugh, Latha Soorya, A. Ting Wang, Eric Hollander, and Evdokia Anagnostou. "Impaired Structural Connectivity of Socio-Emotional Circuits in Autism Spectrum Disorders: A Diffusion Tensor Imaging Study." Edited by Xi-Nian Zuo. *PLoS ONE* 6, no. 11 (November 23, 2011): e28044. https://doi.org/10.1371/journal.pone.0028044.

Ames, Jennifer L., Elizabeth H. Morgan, Morénike Giwa Onaiwu, Yinge Qian, Maria L. Massolo, and Lisa A. Croen. "Racial/Ethnic Differences in Psychiatric and Medical Diagnoses Among Autistic Adults." *Autism in Adulthood: Challenges and Management* 4, no. 4 (December 1, 2022): 290–305. https://doi.org/10.1089/aut.2021.0083.

Anagnostou, Evdokia, Michael G. Aman, Benjamin L. Handen, Kevin B. Sanders, Amy Shui, Jill A. Hollway, Jessica Brian, et al. "Metformin for Treatment of Overweight Induced by

Atypical Antipsychotic Medication in Young People With Autism Spectrum Disorder: A Randomized Clinical Trial." *JAMA Psychiatry* 73, no. 9 (September 1, 2016): 928. https://doi.org/10.1001/jamapsychiatry.2016.1232.

Anagnostou, Evdokia, Latha Soorya, William Chaplin, Jennifer Bartz, Danielle Halpern, Stacey Wasserman, A Ting Wang, et al. "Intranasal Oxytocin versus Placebo in the Treatment of Adults with Autism Spectrum Disorders: A Randomized Controlled Trial." *Molecular Autism* 3, no. 1 (2012): 16. https://doi.org/10.1186/2040-2392-3-16.

Anand, Deepa, Gabriela D. Colpo, Gregory Zeni, Cristian P. Zeni, and Antonio L. Teixeira. "Attention-Deficit/Hyperactivity Disorder And Inflammation: What Does Current Knowledge Tell Us? A Systematic Review." *Frontiers in Psychiatry* 8 (November 9, 2017): 228. https://doi.org/10.3389/fpsyt.2017.00228.

Anavi-Goffer, Sharon, Gemma Baillie, Andrew J. Irving, Jürg Gertsch, Iain R. Greig, Roger G. Pertwee, and Ruth A. Ross. "Modulation of L-α-Lysophosphatidylinositol/GPR55 Mitogen-Activated Protein Kinase (MAPK) Signaling by Cannabinoids." *The Journal of Biological Chemistry* 287, no. 1 (January 2, 2012): 91–104. https://doi.org/10.1074/jbc.M111.296020.

Andalib, S., M.R. Emamhadi, S. Yousefzadeh-Chabok, S.K. Shakouri, P.F. Høilund-Carlsen, M.S. Vafaee, and T.M. Michel. "Maternal SSRI Exposure Increases the Risk of Autistic Offspring: A Meta-Analysis and Systematic Review." *European Psychiatry* 45 (September 2017): 161–66. https://doi.org/10.1016/j.eurpsy.2017.06.001.

Andari, Elissar, Jean-René Duhamel, Tiziana Zalla, Evelyn Herbrecht, Marion Leboyer, and Angela Sirigu. "Promoting Social Behavior with Oxytocin in High-Functioning Autism Spectrum Disorders." *Proceedings of the National Academy of Sciences* 107, no. 9 (March 2, 2010): 4389–94. https://doi.org/10.1073/pnas.0910249107.

Anderson, Brian J. "Paracetamol (Acetaminophen): Mechanisms of Action." *Pediatric Anesthesia* 18, no. 10 (2008): 915–21. https://doi.org/10.1111/j.1460-9592.2008.02764.x.

Anderson, George M., William C. Horne, Diptendu Chatterjee, and Donald J. Cohen. "The Hyperserotonemia of Autisma." *Annals of the New York Academy of Sciences* 600, no. 1 (1990): 331–40. https://doi.org/10.1111/j.1749-6632.1990.tb16893.x.

Anderson, Nathan P., Jamie A. Feldman, David J. Kolko, Paul A. Pilkonis, and Oliver Lindhiem. "National Norms for the Vanderbilt ADHD Diagnostic Parent Rating Scale in Children." *Journal of Pediatric Psychology* 47, no. 6 (July 2022): 652. https://doi.org/10.1093/jpepsy/jsab132.

Andescavage, Nickie N., Adre Du Plessis, Robert McCarter, Ahmed Serag, Iordanis Evangelou, Gilbert Vezina, Richard Robertson, and Catherine Limperopoulos. "Complex Trajectories of Brain Development in the Healthy Human Fetus." *Cerebral Cortex*, October 31, 2016, cercor;bhw306v1. https://doi.org/10.1093/cercor/bhw306.

Anello, Alene, Abraham Reichenberg, Xiaodong Luo, James Schmeidler, Eric Hollander, Christopher J. Smith, Connor M. Puleo, Lauren A. Kryzak, and Jeremy M. Silverman. "Brief Report: Parental Age and the Sex Ratio in Autism." *Journal of Autism and Developmental Disorders* 39, no. 10 (October 2009): 1487–92.

https://doi.org/10.1007/s10803-009-0755-y.

Angelis, Dimitrios, Rashmin C. Savani, Jawahar Jagarapu, June Hu, Phyllis Wan-Huen, and Lina Chalak. "Part I. Mechanisms of Actions and Metabolism of Acetaminophen Related to the Neonatal Brain." *Early Human Development* 159 (August 1, 2021): 105406. https://doi.org/10.1016/j.earlhumdev.2021.105406.

Antar, Laura N., Casara Jean Ferretti, and Eric Hollander. "Immune Response and Inflammation in Autism Spectrum Disorder." *Psychiatric Annals* 42, no. 9 (September 2012): 342–46. https://doi.org/10.3928/00485713-20120906-08.

"Anterior Cingulate Gyrus Volume and Glucose Metabolism in Autistic Disorder." *American Journal of Psychiatry* 154, no. 8 (August 1997): 1047–50. https://doi.org/10.1176/ajp.154.8.1047.

Antshel, Kevin M., and Natalie Russo. "Autism Spectrum Disorders and ADHD: Overlapping Phenomenology, Diagnostic Issues, and Treatment Considerations." *Current Psychiatry Reports* 21, no. 5 (March 22, 2019): 34. https://doi.org/10.1007/s11920-019-1020-5.

Aran, Adi, Maya Eylon, Moria Harel, Lola Polianski, Alina Nemirovski, Sigal Tepper, Aviad Schnapp, Hanoch Cassuto, Nadia Wattad, and Joseph Tam. "Lower Circulating Endocannabinoid Levels in Children with Autism Spectrum Disorder." *Molecular Autism* 10 (January 30, 2019): 2. https://doi.org/10.1186/s13229-019-0256-6.

Arias, Alexis A., Madison M. Rea, Elyse J. Adler, Angela D. Haendel, and Amy Vaughan Van Hecke. "Utilizing the Child Behavior Checklist (CBCL) as an Autism Spectrum Disorder Preliminary Screener and Outcome Measure for the PEERS® Intervention for Autistic Adolescents." *Journal of Autism and Developmental Disorders* 52, no. 5 (May 2022): 2061–74. https://doi.org/10.1007/s10803-021-05103-8.

Arnevik, Espen Ajo, and Sissel Berge Helverschou. "Autism Spectrum Disorder and Co-Occurring Substance Use Disorder – A Systematic Review." *Substance Abuse: Research and Treatment* 10 (January 2016): SART.S39921. https://doi.org/10.4137/SART.S39921.

Arrode-Brusés, Géraldine, and Juan L Brusés. "Maternal Immune Activation by Poly(I:C) Induces Expression of Cytokines IL-1β and IL-13, Chemokine MCP-1 and Colony Stimulating Factor VEGF in Fetal Mouse Brain." *Journal of Neuroinflammation* 9, no. 1 (December 2012): 605. https://doi.org/10.1186/1742-2094-9-83.

Arteche-López, Ana, Maria José Gómez Rodríguez, Maria Teresa Sánchez Calvin, Juan Francisco Quesada-Espinosa, Jose Miguel Lezana Rosales, Carmen Palma Milla, Irene Gómez-Manjón, et al. "Towards a Change in the Diagnostic Algorithm of Autism Spectrum Disorders: Evidence Supporting Whole Exome Sequencing as a First-Tier Test." *Genes* 12, no. 4 (April 12, 2021): 560. https://doi.org/10.3390/genes12040560.

ASEBA. "School-Age (CBCL, TRF, YSR)." Accessed June 22, 2023. https://aseba.org/school-age/.

Ashwood, P, A Enstrom, P Krakowiak, I Hertzpicciotto, R Hansen, L Croen, S Ozonoff, I Pessah, and J Dewater. "Decreased Transforming Growth Factor Beta1 in Autism: A Potential Link between Immune Dysregulation and Impairment in Clinical Behavioral

Outcomes." *Journal of Neuroimmunology* 204, no. 1–2 (November 15, 2008): 149–53. https://doi.org/10.1016/j.jneuroim.2008.07.006.

Ashwood, Paul, Paula Krakowiak, Irva Hertz-Picciotto, Robin Hansen, Isaac N. Pessah, and Judy Van De Water. "Altered T Cell Responses in Children with Autism." *Brain, Behavior, and Immunity* 25, no. 5 (July 2011): 840–49. https://doi.org/10.1016/j.bbi.2010.09.002.

———. "Associations of Impaired Behaviors with Elevated Plasma Chemokines in Autism Spectrum Disorders." *Journal of Neuroimmunology* 232, no. 1–2 (March 2011): 196–99. https://doi.org/10.1016/j.jneuroim.2010.10.025.

Ashwood, Paul, Paula Krakowiak, Irva Hertz-Picciotto, Robin Hansen, Isaac Pessah, and Judy Van de Water. "Elevated Plasma Cytokines in Autism Spectrum Disorders Provide Evidence of Immune Dysfunction and Are Associated with Impaired Behavioral Outcome." *Brain, Behavior, and Immunity* 25, no. 1 (January 2011): 40–45. https://doi.org/10.1016/j.bbi.2010.08.003.

Atladóttir, Hjördís Ó., Marianne G. Pedersen, Poul Thorsen, Preben Bo Mortensen, Bent Deleuran, William W. Eaton, and Erik T. Parner. "Association of Family History of Autoimmune Diseases and Autism Spectrum Disorders." *Pediatrics* 124, no. 2 (August 1, 2009): 687–94. https://doi.org/10.1542/peds.2008-2445.

Atladóttir, Hjördis O., Poul Thorsen, Lars Østergaard, Diana E. Schendel, Sanne Lemcke, Morsi Abdallah, and Erik T. Parner. "Maternal Infection Requiring Hospitalization during Pregnancy and Autism Spectrum Disorders." *Journal of Autism and Developmental Disorders* 40, no. 12 (December 2010): 1423–30. https://doi.org/10.1007/s10803-010-1006-y.

Atladóttir, Hjördis Ósk, Tine Brink Henriksen, Diana E. Schendel, and Erik T. Parner. "Autism After Infection, Febrile Episodes, and Antibiotic Use During Pregnancy: An Exploratory Study." *Pediatrics* 130, no. 6 (December 1, 2012): e1447–54. https://doi.org/10.1542/peds.2012-1107.

Atsem, Stefanie, Juliane Reichenbach, Ramya Potabattula, Marcus Dittrich, Caroline Nava, Christel Depienne, Lena Böhm, et al. "Paternal Age Effects on Sperm FOXK1 and KCNA7 Methylation and Transmission into the next Generation." *Human Molecular Genetics* 25, no. 22 (November 15, 2016): 4996–5005. https://doi.org/10.1093/hmg/ddw328.

Austerman, Joseph. "ADHD and Behavioral Disorders: Assessment, Management, and an Update from DSM-5." *Cleveland Clinic Journal of Medicine* 82, no. 11 suppl 1 (November 2015): S2–7. https://doi.org/10.3949/ccjm.82.s1.01.

"Autism Presentation in Female and Black Populations: Examining the Roles of Identity, Theory, and Systemic Inequalities." Accessed May 15, 2023. https://doi.org/10.1177/13623613221113501.

Autism Research Centre. "Childhood Autism Spectrum Test (CAST)." Accessed June 22, 2023. https://www.autismresearchcentre.com/tests/childhood-autism-spectrum-test-cast/.

Avella-Garcia, Claudia B., Jordi Julvez, Joan Fortuny, Cristina Rebordosa, Raquel García-Esteban, Isolina Riaño Galán, Adonina Tardón, et al. "Acetaminophen Use in Pregnancy and Neurodevelopment: Attention Function and Autism Spectrum Symptoms." *International Journal of Epidemiology* 45, no. 6 (June 28, 2016): dyw115. https://doi.org/10.1093/ije/dyw115.

Ayoub, Samir S. "Paracetamol (Acetaminophen): A Familiar Drug with an Unexplained Mechanism of Action." *Temperature (Austin, Tex.)* 8, no. 4 (2021): 351–71. https://doi.org/10.1080/23328940.2021.1886392.

Bailey, Michael T, Gabriele R Lubach, and Christopher L Coe. "Prenatal Stress Alters Bacterial Colonization of the Gut in Infant Monkeys." *J Pediatr Gastroenterol Nutr* 38, no. 4 (2004).

Baio, John. "Prevalence of Autism Spectrum Disorder Among Children Aged 8 Years — Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2016," n.d.

Baker, Brennan H., Heather H. Burris, Tessa R. Bloomquist, Amélie Boivin, Virginie Gillet, Annie Larouche, Larissa Takser, Jean-Philippe Bellenger, Jean-Charles Pasquier, and Andrea A. Baccarelli. "Association of Prenatal Acetaminophen Exposure Measured in Meconium With Adverse Birth Outcomes in a Canadian Birth Cohort." *Frontiers in Pediatrics* 10 (April 5, 2022): 828089. https://doi.org/10.3389/fped.2022.828089.

Baker, Brennan H, Claudia Lugo-Candelas, Haotian Wu, Hannah E Laue, Amélie Boivin, Virginie Gillet, Natalie Aw, et al. "Association of Prenatal Acetaminophen Exposure Measured in Meconium With Risk of Attention-Deficit/Hyperactivity Disorder Mediated by Frontoparietal Network Brain Connectivity," 2020, 9.

Balasubramanian, Balasubramanian, and Birundha Birundha. "Estimation of Glutathione Level in Second Trimester of Pregnancy without Complications." *Scholars International Journal of Biochemistry* 02, no. 09 (September 30, 2019): 237–39. https://doi.org/10.36348/SIJB.2019.v02i09.003.

Barch, Deanna M. "What Does It Mean to Be Transdiagnostic and How Would We Know?" *American Journal of Psychiatry* 177, no. 5 (May 2020): 370–72. https://doi.org/10.1176/appi.ajp.2020.20030243.

Barde, Y A, D Edgar, and H Thoenen. "Purification of a New Neurotrophic Factor from Mammalian Brain." *The EMBO Journal* 1, no. 5 (1982): 549–53.

Baribeau, Danielle, and Evdokia Anagnostou. "Novel Treatments for Autism Spectrum Disorder Based on Genomics and Systems Biology." *Pharmacology & Therapeutics* 230 (February 2022): 107939. https://doi.org/10.1016/j.pharmthera.2021.107939.

Baron-Cohen, Simon. "Editorial Perspective: Neurodiversity - a Revolutionary Concept for Autism and Psychiatry." *Journal of Child Psychology and Psychiatry* 58, no. 6 (June 2017): 744–47. https://doi.org/10.1111/jcpp.12703.

Barrett, Sarah L., Mirko Uljarević, Emma K. Baker, Amanda L. Richdale, Catherine R. G. Jones, and Susan R. Leekam. "The Adult Repetitive Behaviours Questionnaire-2 (RBQ-2A): A Self-Report Measure of Restricted and Repetitive Behaviours." *Journal of Autism and*

Developmental Disorders 45, no. 11 (November 2015): 3680–92. https://doi.org/10.1007/s10803-015-2514-6.

Barrett, Sarah L., Mirko Uljarević, Catherine R. G. Jones, and Susan R. Leekam. “Assessing Subtypes of Restricted and Repetitive Behaviour Using the Adult Repetitive Behaviour Questionnaire-2 in Autistic Adults.” *Molecular Autism* 9, no. 1 (December 2018): 58. https://doi.org/10.1186/s13229-018-0242-4.

Basavarajappa, Balapal S., Ralph A Nixon, and Ottavio Arancio. “Endocannabinoid System: Emerging Role from Neurodevelopment to Neurodegeneration.” *Mini Reviews in Medicinal Chemistry* 9, no. 4 (April 2009): 448–62.

Battista, Natalia, Monia Di Tommaso, Monica Bari, and Mauro Maccarrone. “The Endocannabinoid System: An Overview.” *Frontiers in Behavioral Neuroscience* 6 (March 14, 2012): 9. https://doi.org/10.3389/fnbeh.2012.00009.

Bauer, Ann Z, and David Kriebel. “Prenatal and Perinatal Analgesic Exposure and Autism: An Ecological Link.” *Environmental Health* 12, no. 1 (December 2013): 41. https://doi.org/10.1186/1476-069X-12-41.

Bauer, Ann Z., David Kriebel, Martha R. Herbert, Carl-Gustaf Bornehag, and Shanna H. Swan. “Prenatal Paracetamol Exposure and Child Neurodevelopment: A Review.” *Hormones and Behavior* 101 (May 2018): 125–47. https://doi.org/10.1016/j.yhbeh.2018.01.003.

Bauer, Ann Z., Shanna H. Swan, David Kriebel, Zeyan Liew, Hugh S. Taylor, Carl-Gustaf Bornehag, Anderson M. Andrade, et al. “Paracetamol Use during Pregnancy—A Call for Precautionary Action.” *Nature Reviews Endocrinology* 17, no. 12 (December 2021): 757–66. https://doi.org/10.1038/s41574-021-00553-7.

———. “Reply to ‘Paracetamol Use in Pregnancy—Neglecting Context Promotes Misinterpretation.’” *Nature Reviews Endocrinology* 18, no. 6 (June 2022): 386–386. https://doi.org/10.1038/s41574-022-00657-8.

Bauer, Ann Z., Shanna H. Swan, David Kriebel, Zeyan Liew, Hugh S. Taylor, Carl-Gustaf Bornehag, Anderson M. Andrade, et al. “Reply to ‘Paracetamol Use in Pregnancy — Caution over Causal Inference from Available Data’; ‘Handle with Care — Interpretation, Synthesis and Dissemination of Data on Paracetamol in Pregnancy.’” *Nature Reviews Endocrinology* 18, no. 3 (March 2022): 192–192. https://doi.org/10.1038/s41574-021-00610-1.

Bauman, M D, A-M Iosif, P Ashwood, D Braunschweig, A Lee, C M Schumann, J Van De Water, and D G Amaral. “Maternal Antibodies from Mothers of Children with Autism Alter Brain Growth and Social Behavior Development in the Rhesus Monkey.” *Translational Psychiatry* 3, no. 7 (July 9, 2013): e278–e278. https://doi.org/10.1038/tp.2013.47.

Bauman, Melissa D., Ana-Maria Iosif, Stephen E.P. Smith, Catherine Bregere, David G. Amaral, and Paul H. Patterson. “Activation of the Maternal Immune System During Pregnancy Alters Behavioral Development of Rhesus Monkey Offspring.” *Biological Psychiatry* 75, no. 4 (February 2014): 332–41. https://doi.org/10.1016/j.biopsych.2013.06.025.

Beck, Kelly B., Caitlin M. Conner, Susan W. White, and Carla A. Mazefsky. "Mindfulness 'Here and Now': Strategies for Helping Adolescents With Autism." *Journal of the American Academy of Child and Adolescent Psychiatry* 59, no. 10 (October 2020): 1125–27. https://doi.org/10.1016/j.jaac.2020.07.004.

Beltramo, Massimiliano, Fernando Rodríguez De Fonseca, Miguel Navarro, Antonio Calignano, Miguel Angel Gorriti, Gerasimos Grammatikopoulos, Adolfo G. Sadile, Andrea Giuffrida, and Daniele Piomelli. "Reversal of Dopamine D $_2$ Receptor Responses by an Anandamide Transport Inhibitor." *The Journal of Neuroscience* 20, no. 9 (May 1, 2000): 3401–7. https://doi.org/10.1523/JNEUROSCI.20-09-03401.2000.

Ben-Ari, Yehezkel, and Eric Lemonnier. "Using Bumetanide to Treat Autism Appears Promising but Further Clinical Trials Are Needed to Confirm This Approach." *Acta Paediatrica* 110, no. 5 (May 2021): 1395–97. https://doi.org/10.1111/apa.15747.

Bent, Stephen, Kiah Bertoglio, Paul Ashwood, Edward Nemeth, and Robert L. Hendren. "Brief Report: Hyperbaric Oxygen Therapy (HBOT) in Children with Autism Spectrum Disorder: A Clinical Trial." *Journal of Autism and Developmental Disorders* 42, no. 6 (June 2012): 1127–32. https://doi.org/10.1007/s10803-011-1337-3.

Bernaerts, Sylvie, Bart Boets, Guy Bosmans, Jean Steyaert, and Kaat Alaerts. "Behavioral Effects of Multiple-Dose Oxytocin Treatment in Autism: A Randomized, Placebo-Controlled Trial with Long-Term Follow-Up." *Molecular Autism* 11, no. 1 (December 2020): 6. https://doi.org/10.1186/s13229-020-0313-1.

Bernardi, Silvia, Evdokia Anagnostou, Jun Shen, Alexander Kolevzon, Joseph D. Buxbaum, Eric Hollander, Patrick R. Hof, and Jin Fan. "In Vivo 1H-Magnetic Resonance Spectroscopy Study of the Attentional Networks in Autism." *Brain Research* 1380 (March 2011): 198–205. https://doi.org/10.1016/j.brainres.2010.12.057.

Bernardi, Silvia, Samuele Cortese, Mary Solanto, Eric Hollander, and Stefano Pallanti. "Bipolar Disorder and Comorbid Attention Deficit Hyperactivity Disorder. A Distinct Clinical Phenotype? Clinical Characteristics and Temperamental Traits [1]." *The World Journal of Biological Psychiatry* 11, no. 4 (January 2010): 656–66. https://doi.org/10.3109/15622971003653238.

Bernstein, Bradley E., Alexander Meissner, and Eric S. Lander. "The Mammalian Epigenome." *Cell* 128, no. 4 (February 2007): 669–81. https://doi.org/10.1016/j.cell.2007.01.033.

Bertelsen, Elin Næs, Janne Tidselbak Larsen, Liselotte Petersen, Jakob Christensen, and Søren Dalsgaard. "Childhood Epilepsy, Febrile Seizures, and Subsequent Risk of ADHD." *Pediatrics* 138, no. 2 (August 2016): e20154654. https://doi.org/10.1542/peds.2015-4654.

Bertoldi, Andréa D., Sheryl L. Rifas-Shiman, Alexandra Crispim Boing, Tatiane Silva Dal Pizzol, Vanessa Iribarrem Avena Miranda, Marysabel Pinto Telis Silveira, Mariângela Freitas Silveira, et al. "Associations of Acetaminophen Use during Pregnancy and the First Year of Life with Neurodevelopment in Early Childhood." *Paediatric and Perinatal Epidemiology* 34, no. 3 (May 2020): 267–77. https://doi.org/10.1111/ppe.12632.

Bertolini, Alfio, Anna Ferrari, Alessandra Ottani, Simona Guerzoni, Raffaella Tacchi, and Sheila Leone. "Paracetamol: New Vistas of an Old Drug." *CNS Drug Reviews* 12, no. 3–4

(September 2006): 250–75. https://doi.org/10.1111/j.1527-3458.2006.00250.x.

Biederman, Joseph, Sarah W. Ball, Michael C. Monuteaux, Eric Mick, Thomas J. Spencer, Michelle McCREARY, Michelle Cote, and Stephen V. Faraone. “New Insights into the Comorbidity between ADHD and Major Depression in Adolescent and Young Adult Females.” *Journal of the American Academy of Child and Adolescent Psychiatry* 47, no. 4 (April 2008): 426–34. https://doi.org/10.1097/CHI.0b013e31816429d3.

Biederman, Joseph, Maura DiSalvo, Carrie Vaudreuil, Janet Wozniak, Mai Uchida, K. Yvonne Woodworth, Allison Green, Abigail Farrell, and Stephen V. Faraone. “The Child Behavior Checklist Can Aid in Characterizing Suspected Comorbid Psychopathology in Clinically Referred Youth with ADHD.” *Journal of Psychiatric Research* 138 (June 2021): 477–84. https://doi.org/10.1016/j.jpsychires.2021.04.028.

Biederman, Joseph, and Stephen V Faraone. “Attention-Deficit Hyperactivity Disorder” 366 (2005).

Bishop, Somer L., Jennifer Richler, and Catherine Lord. “Association Between Restricted and Repetitive Behaviors and Nonverbal IQ in Children with Autism Spectrum Disorders.” *Child Neuropsychology* 12, no. 4–5 (October 2006): 247–67. https://doi.org/10.1080/09297040600630288.

Bitanihirwe, Byron KY, Daria Peleg-Raibstein, Forouhar Mouttet, Joram Feldon, and Urs Meyer. “Late Prenatal Immune Activation in Mice Leads to Behavioral and Neurochemical Abnormalities Relevant to the Negative Symptoms of Schizophrenia.” *Neuropsychopharmacology* 35, no. 12 (November 2010): 2462–78. https://doi.org/10.1038/npp.2010.129.

Bitsko, Rebecca H., Angelika H. Claussen, Jesse Lichstein, Lindsey I. Black, Sherry Everett Jones, Melissa L. Danielson, Jennifer M. Hoenig, et al. “Mental Health Surveillance Among Children — United States, 2013–2019.” *MMWR Supplements* 71, no. 2 (February 25, 2022): 1–42. https://doi.org/10.15585/mmwr.su7102a1.

Bjørklund, Geir, Nagwa A. Meguid, Mona A. El-Bana, Alexey A. Tinkov, Khaled Saad, Maryam Dadar, Maha Hemimi, et al. “Oxidative Stress in Autism Spectrum Disorder.” *Molecular Neurobiology* 57, no. 5 (May 1, 2020): 2314–32. https://doi.org/10.1007/s12035-019-01742-2.

Black, Ronald A., and D. Ashley Hill. “Over-the-Counter Medications in Pregnancy.” *American Family Physician* 67, no. 12 (June 15, 2003): 2517–24.

Blecharz-Klin, Kamilla, Ilona Joniec-Maciejak, Katarzyna Jawna, Justyna Pyrzanowska, Agnieszka Piechal, Adriana Wawer, and Ewa Widy-Tyszkiewicz. “Developmental Exposure to Paracetamol Causes Biochemical Alterations in Medulla Oblongata.” *Environmental Toxicology and Pharmacology* 40, no. 2 (September 2015): 369–74. https://doi.org/10.1016/j.etap.2015.07.001.

———. “Effect of Prenatal and Early Life Paracetamol Exposure on the Level of Neurotransmitters in Rats—Focus on the Spinal Cord.” *International Journal of Developmental Neuroscience* 47, no. Part_B (2015): 133–39. https://doi.org/10.1016/j.ijdevneu.2015.09.002.

Blecharz-Klin, Kamilla, Ilona Joniec-Maciejak, Katarzyna Jawna-Zboińska, Justyna Pyrzanowska, Agnieszka Piechal, Adriana Wawer, and Ewa Widy-Tyszkiewicz. “Cerebellar Level of Neurotransmitters in Rats Exposed to Paracetamol during Development.” *Pharmacological Reports: PR* 68, no. 6 (December 2016): 1159–64. https://doi.org/10.1016/j.pharep.2016.06.005.

Blecharz-Klin, Kamilla, Agnieszka Piechal, Katarzyna Jawna-Zboińska, Justyna Pyrzanowska, Adriana Wawer, Ilona Joniec-Maciejak, and Ewa Widy-Tyszkiewicz. “Paracetamol - Effect of Early Exposure on Neurotransmission, Spatial Memory and Motor Performance in Rats.” *Behavioural Brain Research* 323 (April 14, 2017): 162–71. https://doi.org/10.1016/j.bbr.2017.01.051.

Boccuto, Luigi, Maria Lauri, Sara M Sarasua, Cindy D Skinner, Daniela Buccella, Alka Dwivedi, Daniela Orteschi, et al. “Prevalence of SHANK3 Variants in Patients with Different Subtypes of Autism Spectrum Disorders.” *European Journal of Human Genetics* 21, no. 3 (March 2013): 310–16. https://doi.org/10.1038/ejhg.2012.175.

Bodfish, James W, Frank J Symons, Dawn E Parker, and Mark H Lewis. “Varieties of Repetitive Behavior in Autism: Comparisons to Mental Retardation,” n.d.

Boedhoe, Premika S. W., Daan van Rooij, Martine Hoogman, Jos W. R. Twisk, Lianne Schmaal, Yoshinari Abe, Pino Alonso, et al. “Subcortical Brain Volume, Regional Cortical Thickness, and Cortical Surface Area Across Disorders: Findings From the ENIGMA ADHD, ASD, and OCD Working Groups.” *The American Journal of Psychiatry* 177, no. 9 (September 1, 2020): 834–43. https://doi.org/10.1176/appi.ajp.2020.19030331.

Bolognani, Federico, Marta Del Valle Rubido, Lisa Squassante, Christoph Wandel, Michael Derks, Lorraine Murtagh, Jeff Sevigny, Omar Khwaja, Daniel Umbricht, and Paulo Fontoura. “A Phase 2 Clinical Trial of a Vasopressin V1a Receptor Antagonist Shows Improved Adaptive Behaviors in Men with Autism Spectrum Disorder.” *Science Translational Medicine* 11, no. 491 (May 8, 2019): eaat7838. https://doi.org/10.1126/scitranslmed.aat7838.

Bornehag, C.-G., A. Reichenberg, M. Unenge Hallerback, S. Wikstrom, H.M. Koch, B.A. Jonsson, and S.H. Swan. “Prenatal Exposure to Acetaminophen and Children’s Language Development at 30 Months.” *European Psychiatry* 51 (June 2018): 98–103. https://doi.org/10.1016/j.eurpsy.2017.10.007.

Borue, Xenia, Carla Mazefsky, Brian T. Rooks, Michael Strober, Martin B. Keller, Heather Hower, Shirley Yen, et al. “Longitudinal Course of Bipolar Disorder in Youth With High-Functioning Autism Spectrum Disorder.” *Journal of the American Academy of Child & Adolescent Psychiatry* 55, no. 12 (December 2016): 1064-1072.e6. https://doi.org/10.1016/j.jaac.2016.08.011.

Bos, Dienke J., Melanie R. Silverman, Eliana L. Ajodan, Cynthia Martin, Benjamin M. Silver, Gijs J. Brouwer, Adriana Di Martino, and Rebecca M. Jones. “Rigidity Coincides with Reduced Cognitive Control to Affective Cues in Children with Autism.” *Journal of Abnormal Psychology* 128, no. 5 (July 2019): 431–41. https://doi.org/10.1037/abn0000423.

Bourreau, Yannig, Sylvie Roux, Marie Gomot, Frédérique Bonnet-Brilhault, and Catherine Barthélémy. "Validation of the Repetitive and Restricted Behaviour Scale in Autism Spectrum Disorders." *European Child & Adolescent Psychiatry* 18, no. 11 (November 2009): 675–82. https://doi.org/10.1007/s00787-009-0028-5.

Bowden, Virginia M. "The Journal of Neuropsychiatry and Clinical Neurosciences." *JAMA: The Journal of the American Medical Association* 268, no. 11 (September 16, 1992): 1473. https://doi.org/10.1001/jama.1992.03490110111047.

Bowers, Katherine, Qing Li, Joseph Bressler, Dimitrios Avramopoulos, Craig Newschaffer, and M. Daniele Fallin. "Glutathione Pathway Gene Variation and Risk of Autism Spectrum Disorders." *Journal of Neurodevelopmental Disorders* 3, no. 2 (June 2011): 132–43. https://doi.org/10.1007/s11689-011-9077-4.

Bradshaw, Jessica, Scott Gillespie, Courtney McCracken, Bryan H. King, James T. McCracken, Cynthia R. Johnson, Luc Lecavalier, et al. "Predictors of Caregiver Strain for Parents of Children with Autism Spectrum Disorder." *Journal of Autism and Developmental Disorders* 51, no. 9 (September 2021): 3039–49. https://doi.org/10.1007/s10803-020-04625-x.

Bradstreet, James Jeffrey, Emar Vogelaar, and Lynda Thyer. "Initial Observations of Elevated Alpha-N-Acetylgalactosaminidase Activity Associated with Autism and Observed Reductions from Gc Protein--Macrophage Activating Factor Injections." *Autism Insights*, December 10, 2012, 31–39. https://doi.org/10.4137/AUI.S10485.

Brandlistuen, R. E., E. Ystrom, I. Nulman, G. Koren, and H. Nordeng. "Prenatal Paracetamol Exposure and Child Neurodevelopment: A Sibling-Controlled Cohort Study." *International Journal of Epidemiology* 42, no. 6 (December 1, 2013): 1702–13. https://doi.org/10.1093/ije/dyt183.

Brandlistuen, Ragnhild Eek, Eivind Ystrom, Irena Nulman, Gideon Koren, and Hedvig Nordeng. "Annotations and Reflections: Response to "Pregnancy and Paracetamol: Methodological Considerations on the Study of Associations between In Utero Exposure to Drugs and Childhood Ne." *Basic & Clinical Pharmacology & Toxicology* 116, no. 1 (January 2015): 6–8. https://doi.org/10.1111/bcpt.12339.

Braunschweig, D, P Krakowiak, P Duncanson, R Boyce, R L Hansen, P Ashwood, I Hertz-Picciotto, I N Pessah, and J Van De Water. "Autism-Specific Maternal Autoantibodies Recognize Critical Proteins in Developing Brain." *Translational Psychiatry* 3, no. 7 (July 9, 2013): e277–e277. https://doi.org/10.1038/tp.2013.50.

Braunschweig, Daniel, Paul Duncanson, Robert Boyce, Robin Hansen, Paul Ashwood, Isaac N. Pessah, Irva Hertz-Picciotto, and Judy Van De Water. "Behavioral Correlates of Maternal Antibody Status Among Children with Autism." *Journal of Autism and Developmental Disorders* 42, no. 7 (July 2012): 1435–45. https://doi.org/10.1007/s10803-011-1378-7.

Breaux, Rosanna P., Shayl F. Griffith, and Elizabeth A. Harvey. "Preschool Neuropsychological Measures as Predictors of Later Attention Deficit Hyperactivity Disorder." *Journal of Abnormal Child Psychology* 44, no. 8 (November 2016): 1455–71. https://doi.org/10.1007/s10802-016-0140-1.

Breece, Elizabeth, Brian Paciotti, Christine Wu Nordahl, Sally Ozonoff, Judy A. Van De Water, Sally J. Rogers, David Amaral, and Paul Ashwood. "Myeloid Dendritic Cells Frequencies Are Increased in Children with Autism Spectrum Disorder and Associated with Amygdala Volume and Repetitive Behaviors." *Brain, Behavior, and Immunity* 31 (July 2013): 69–75. https://doi.org/10.1016/j.bbi.2012.10.006.

Brewer, Rebecca, Federica Biotti, Caroline Catmur, Clare Press, Francesca Happé, Richard Cook, and Geoffrey Bird. "Can Neurotypical Individuals Read Autistic Facial Expressions? Atypical Production of Emotional Facial Expressions in Autism Spectrum Disorders: Expression Production in Autism." *Autism Research* 9, no. 2 (February 2016): 262–71. https://doi.org/10.1002/aur.1508.

Brigandi, Sarah A., Hong Shao, Steven Y. Qian, Yiping Shen, Bai-Lin Wu, and Jing X. Kang. "Autistic Children Exhibit Decreased Levels of Essential Fatty Acids in Red Blood Cells." *International Journal of Molecular Sciences* 16, no. 5 (May 4, 2015): 10061–76. https://doi.org/10.3390/ijms160510061.

"Brigg's Drugs in Pregnancy and Lactation: A Reference Guide to Fetal and Neonatal Risk." Accessed June 22, 2023. https://www.wolterskluwer.com/en/solutions/ovid/briggs-drugs-in-pregnancy-and-lactation-a-reference-guide-to-fetal-and-neonatal-risk-730.

Brigida, Anna Lisa, Stephen Schultz, Mariana Cascone, Nicola Antonucci, and Dario Siniscalco. "Endocannabinod Signal Dysregulation in Autism Spectrum Disorders: A Correlation Link between Inflammatory State and Neuro-Immune Alterations." *International Journal of Molecular Sciences* 18, no. 7 (July 3, 2017): 1425. https://doi.org/10.3390/ijms18071425.

Brikell, Isabell, Laura Ghirardi, Brian M. D'Onofrio, David W. Dunn, Catarina Almqvist, Søren Dalsgaard, Ralf Kuja-Halkola, and Henrik Larsson. "Familial Liability to Epilepsy and Attention-Deficit/Hyperactivity Disorder: A Nationwide Cohort Study." *Biological Psychiatry* 83, no. 2 (January 15, 2018): 173–80. https://doi.org/10.1016/j.biopsych.2017.08.006.

Bronson, Stefanie L., and Tracy L. Bale. "Prenatal Stress-Induced Increases in Placental Inflammation and Offspring Hyperactivity Are Male-Specific and Ameliorated by Maternal Antiinflammatory Treatment." *Endocrinology* 155, no. 7 (July 1, 2014): 2635–46. https://doi.org/10.1210/en.2014-1040.

Brune, K., B. Renner, and G. Tiegs. "Acetaminophen/Paracetamol: A History of Errors, Failures and False Decisions." *European Journal of Pain (London, England)* 19, no. 7 (August 2015): 953–65. https://doi.org/10.1002/ejp.621.

Brunkhorst-Kanaan, Nathalie, Sandra Trautmann, Yannick Schreiber, Dominique Thomas, Sarah Kittel-Schneider, Robert Gurke, Gerd Geisslinger, Andreas Reif, and Irmgard Tegeder. "Sphingolipid and Endocannabinoid Profiles in Adult Attention Deficit Hyperactivity Disorder." *Biomedicines* 9, no. 9 (September 6, 2021): 1173. https://doi.org/10.3390/biomedicines9091173.

Bryn, V., B. Halvorsen, T. Ueland, J. Isaksen, K. Kolkova, K. Ravn, and O. H. Skjeldal. "Brain Derived Neurotrophic Factor (BDNF) and Autism Spectrum Disorders (ASD) in

Childhood." *European Journal of Paediatric Neurology: EJPN: Official Journal of the European Paediatric Neurology Society* 19, no. 4 (July 2015): 411–14. https://doi.org/10.1016/j.ejpn.2015.03.005.

Buchmayer, Susanne, Stefan Johansson, Anna Johansson, Christina M. Hultman, Pär Sparén, and Sven Cnattingius. "Can Association between Preterm Birth and Autism Be Explained by Maternal or Neonatal Morbidity?" *Pediatrics* 124, no. 5 (November 2009): e817-825. https://doi.org/10.1542/peds.2008-3582.

Buchsbaum, Monte S., Eric Hollander, M. Mehmet Haznedar, Cheuk Tang, Jacqueline Spiegel-Cohen, Tse-Chung Wei, Andrea Solimando, et al. "Effect of Fluoxetine on Regional Cerebral Metabolism in Autistic Spectrum Disorders: A Pilot Study." *The International Journal of Neuropsychopharmacology* 4, no. 02 (June 2001). https://doi.org/10.1017/S1461145701002280.

Bührer, Christoph, Stefanie Endesfelder, Till Scheuer, and Thomas Schmitz. "Paracetamol (Acetaminophen) and the Developing Brain." *International Journal of Molecular Sciences* 22, no. 20 (January 2021): 11156. https://doi.org/10.3390/ijms222011156.

Burns, Amy, Mandy Irvine, and Kate Woodcock. "Self-Focused Attention and Depressive Symptoms in Adults with Autistic Spectrum Disorder (ASD)." *Journal of Autism and Developmental Disorders* 49, no. 2 (February 2019): 692–703. https://doi.org/10.1007/s10803-018-3732-5.

Butterworth, Roger F. "Hepatic Encephalopathy: A Central Neuroinflammatory Disorder?" *Hepatology (Baltimore, Md.)* 53, no. 4 (April 2011): 1372–76. https://doi.org/10.1002/hep.24228.

Buxbaum, J D, J Silverman, M Keddache, C J Smith, E Hollander, N Ramoz, and J G Reichert. "Linkage Analysis for Autism in a Subset Families with Obsessive–Compulsive Behaviors: Evidence for an Autism Susceptibility Gene on Chromosome 1 and Further Support for Susceptibility Genes on Chromosome 6 and 19." *Molecular Psychiatry* 9, no. 2 (February 1, 2004): 144–50. https://doi.org/10.1038/sj.mp.4001465.

Buxbaum, Joseph D., Guiqing Cai, Pauline Chaste, Gudrun Nygren, Juliet Goldsmith, Jennifer Reichert, Henrik Anckarsäter, et al. "Mutation Screening of ThePTEN Gene in Patients with Autism Spectrum Disorders and Macrocephaly." *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* 144B, no. 4 (June 5, 2007): 484–91. https://doi.org/10.1002/ajmg.b.30493.

Buxbaum, Joseph D, Guiqing Cai, Gudrun Nygren, Pauline Chaste, Richard Delorme, Juliet Goldsmith, Maria Råstam, et al. "Mutation Analysis of the NSD1 Gene in Patients with Autism Spectrum Disorders and Macrocephaly." *BMC Medical Genetics* 8, no. 1 (December 2007): 68. https://doi.org/10.1186/1471-2350-8-68.

Buxbaum, Joseph D., Jeremy M. Silverman, Christopher J. Smith, Mario Kilifarski, Jennifer Reichert, Eric Hollander, Brian A. Lawlor, Michael Fitzgerald, David A. Greenberg, and Kenneth L. Davis. "Evidence for a Susceptibility Gene for Autism on Chromosome 2 and for Genetic Heterogeneity." *The American Journal of Human Genetics* 68, no. 6 (June 2001): 1514–20. https://doi.org/10.1086/320588.

Cabral, Guy A., Thomas J. Rogers, and Aron H. Lichtman. "Turning Over a New Leaf: Cannabinoid and Endocannabinoid Modulation of Immune Function." *Journal of Neuroimmune Pharmacology: The Official Journal of the Society on NeuroImmune Pharmacology* 10, no. 2 (June 2015): 193–203. https://doi.org/10.1007/s11481-015-9615-z.

Camm-Crosbie, Louise, Louise Bradley, Rebecca Shaw, Simon Baron-Cohen, and Sarah Cassidy. "'People like Me Don't Get Support': Autistic Adults' Experiences of Support and Treatment for Mental Health Difficulties, Self-Injury and Suicidality." *Autism* 23, no. 6 (August 2019): 1431–41. https://doi.org/10.1177/1362361318816053.

Campbell, Kyle A., Ruby Hickman, Margaret Daniele Fallin, and Kelly M. Bakulski. "Prenatal Exposure to Metals and Autism Spectrum Disorder: Current Status and Future Directions." *Current Opinion in Toxicology* 26 (June 1, 2021): 39–48. https://doi.org/10.1016/j.cotox.2021.04.001.

———. "Prenatal Exposure to Metals and Autism Spectrum Disorder: Current Status and Future Directions." *Current Opinion in Toxicology* 26 (June 1, 2021): 39–48. https://doi.org/10.1016/j.cotox.2021.04.001.

Cao, Xinyi, Ping Lin, Ping Jiang, and Chunbo Li. "Characteristics of the Gastrointestinal Microbiome in Children with Autism Spectrum Disorder: A Systematic Review" 25, no. 6 (2013).

Careaga, Milo, Jared Schwartzer, and Paul Ashwood. "Inflammatory Profiles in the BTBR Mouse: How Relevant Are They to Autism Spectrum Disorders?" *Brain, Behavior, and Immunity* 43 (January 2015): 11–16. https://doi.org/10.1016/j.bbi.2014.06.006.

Careaga, Milo, Judy Van de Water, and Paul Ashwood. "Immune Dysfunction in Autism: A Pathway to Treatment." *Neurotherapeutics* 7, no. 3 (July 2010): 283–92. https://doi.org/10.1016/j.nurt.2010.05.003.

Carey, Meghan E., Juliette Rando, Stepan Melnyk, S. Jill James, Nathaniel Snyder, Carolyn Salafia, Lisa A. Croen, et al. "Examining Associations between Prenatal Biomarkers of Oxidative Stress and ASD-Related Outcomes Using Quantile Regression." *Journal of Autism and Developmental Disorders*, June 9, 2022. https://doi.org/10.1007/s10803-022-05625-9.

Cariveau, Tom, Courtney E. McCracken, Jessica Bradshaw, Valentina Postorino, M. Alice Shillingsburg, Christopher J. McDougle, Michael G. Aman, et al. "Gender Differences in Treatment-Seeking Youth with Autism Spectrum Disorder." *Journal of Child and Family Studies* 30, no. 3 (March 2021): 784–92. https://doi.org/10.1007/s10826-021-01905-7.

Carter, C.J., and R.A. Blizard. "Autism Genes Are Selectively Targeted by Environmental Pollutants Including Pesticides, Heavy Metals, Bisphenol A, Phthalates and Many Others in Food, Cosmetics or Household Products." *Neurochemistry International* 101 (December 2016): 83–109. https://doi.org/10.1016/j.neuint.2016.10.011.

Casanova, Manuel F. "The Neuropathology of Autism." *Brain Pathology* 17, no. 4 (October 4, 2007): 422–33. https://doi.org/10.1111/j.1750-3639.2007.00100.x.

Casanova, Manuel F., Daniel Buxhoeveden, and Juan Gomez. "Disruption in the Inhibitory Architecture of the Cell Minicolumn: Implications for Autisim." *The Neuroscientist* 9, no. 6 (December 1, 2003): 496–507. https://doi.org/10.1177/1073858403253552.

———. "Disruption in the Inhibitory Architecture of the Cell Minicolumn: Implications for Autism." *The Neuroscientist: A Review Journal Bringing Neurobiology, Neurology and Psychiatry* 9, no. 6 (December 2003): 496–507. https://doi.org/10.1177/1073858403253552.

Casanova, Manuel F., and Christopher R. Tillquist. "Encephalization, Emergent Properties, and Psychiatry: A Minicolumnar Perspective." *The Neuroscientist* 14, no. 1 (February 1, 2008): 101–18. https://doi.org/10.1177/1073858407309091.

Cassidy, S. A., K. Gould, E. Townsend, M. Pelton, A. E. Robertson, and J. Rodgers. "Is Camouflaging Autistic Traits Associated with Suicidal Thoughts and Behaviours? Expanding the Interpersonal Psychological Theory of Suicide in an Undergraduate Student Sample." *Journal of Autism and Developmental Disorders* 50, no. 10 (October 2020): 3638–48. https://doi.org/10.1007/s10803-019-04323-3.

Cassidy, Sarah A., Louise Bradley, Heather Cogger-Ward, Rebecca Shaw, Erica Bowen, Magdalena Glod, Simon Baron-Cohen, and Jacqui Rodgers. "Measurement Properties of the Suicidal Behaviour Questionnaire-Revised in Autistic Adults." *Journal of Autism and Developmental Disorders* 50, no. 10 (October 2020): 3477–88. https://doi.org/10.1007/s10803-020-04431-5.

Cassidy, Sarah, Louise Bradley, Rebecca Shaw, and Simon Baron-Cohen. "Risk Markers for Suicidality in Autistic Adults." *Molecular Autism* 9, no. 1 (December 2018): 42. https://doi.org/10.1186/s13229-018-0226-4.

Cassidy, Sarah, Paul Bradley, Janine Robinson, Carrie Allison, Meghan McHugh, and Simon Baron-Cohen. "Suicidal Ideation and Suicide Plans or Attempts in Adults with Asperger's Syndrome Attending a Specialist Diagnostic Clinic: A Clinical Cohort Study." *The Lancet Psychiatry* 1, no. 2 (July 2014): 142–47. https://doi.org/10.1016/S2215-0366(14)70248-2.

Cassidy, Sarah, and Jacqui Rodgers. "Understanding and Prevention of Suicide in Autism." *The Lancet Psychiatry* 4, no. 6 (June 2017): e11. https://doi.org/10.1016/S2215-0366(17)30162-1.

Catalá-López, Ferrán, Brian Hutton, Amparo Núñez-Beltrán, Matthew J. Page, Manuel Ridao, Diego Macías Saint-Gerons, Miguel A. Catalá, Rafael Tabarés-Seisdedos, and David Moher. "The Pharmacological and Non-Pharmacological Treatment of Attention Deficit Hyperactivity Disorder in Children and Adolescents: A Systematic Review with Network Meta-Analyses of Randomised Trials." *PLoS ONE* 12, no. 7 (July 12, 2017): e0180355. https://doi.org/10.1371/journal.pone.0180355.

Caye, Arthur, James M. Swanson, David Coghill, and Luis Augusto Rohde. "Treatment Strategies for ADHD: An Evidence-Based Guide to Select Optimal Treatment." *Molecular Psychiatry* 24, no. 3 (March 2019): 390–408. https://doi.org/10.1038/s41380-018-0116-3.

Cendejas-Hernandez, Jasmine, Joshua T. Sarafian, Victoria G. Lawton, Antara Palkar, Lauren G. Anderson, Vincent Larivière, and William Parker. “Paracetamol (Acetaminophen) Use in Infants and Children Was Never Shown to Be Safe for Neurodevelopment: A Systematic Review with Citation Tracking.” *European Journal of Pediatrics* 181, no. 5 (May 2022): 1835–57. https://doi.org/10.1007/s00431-022-04407-w.

Centers for Disease Control and Prevention. “Data and Statistics About ADHD | CDC,” June 8, 2022. https://www.cdc.gov/ncbddd/adhd/data.html.

Cervantes, Paige, Sarah Kuriakose, Lauren Donnelly, Beryl Filton, Mollie Marr, Eugene Okparaeke, Katherine Voorheis, Jennifer Havens, and Sarah Horwitz. “Sustainability of a Care Pathway for Children and Adolescents with Autism Spectrum Disorder on an Inpatient Psychiatric Service.” *Journal of Autism and Developmental Disorders* 49, no. 8 (August 2019): 3173–80. https://doi.org/10.1007/s10803-019-04029-6.

Chakrabarti, Bhismadev, and Simon Baron-Cohen. “Variation in the Human Cannabinoid Receptor CNR1 Gene Modulates Gaze Duration for Happy Faces.” *Molecular Autism* 2 (June 29, 2011): 10. https://doi.org/10.1186/2040-2392-2-10.

Chakrabarti, Bhismadev, Antonio Persico, Natalia Battista, and Mauro Maccarrone. “Endocannabinoid Signaling in Autism.” *Neurotherapeutics* 12, no. 4 (October 2015): 837–47. https://doi.org/10.1007/s13311-015-0371-9.

Chaliha, Devahuti, Matthew Albrecht, Mauro Vaccarezza, Ryu Takechi, Virginie Lam, Hani Al-Salami, and John Mamo. “A Systematic Review of the Valproic-Acid-Induced Rodent Model of Autism.” *Developmental Neuroscience* 42, no. 1 (2020): 12–48. https://doi.org/10.1159/000509109.

Chang, Jane Pei-Chen, Kuan-Pin Su, Valeria Mondelli, and Carmine M. Pariante. “Cortisol and Inflammatory Biomarker Levels in Youths with Attention Deficit Hyperactivity Disorder (ADHD): Evidence from a Systematic Review with Meta-Analysis.” *Translational Psychiatry* 11 (August 19, 2021): 430. https://doi.org/10.1038/s41398-021-01550-0.

Chang, Ling-Yin, Mei-Yeh Wang, and Pei-Shan Tsai. “Diagnostic Accuracy of Rating Scales for Attention-Deficit/Hyperactivity Disorder: A Meta-Analysis.” *Pediatrics* 137, no. 3 (March 1, 2016): e20152749. https://doi.org/10.1542/peds.2015-2749.

Channing, Jonathan, Mary Mitchell, and Samuele Cortese. “Lurasidone in Children and Adolescents: Systematic Review and Case Report.” *Journal of Child and Adolescent Psychopharmacology* 28, no. 7 (September 2018): 428–36. https://doi.org/10.1089/cap.2018.0046.

Charman, Tony, Gillian Baird, Emily Simonoff, Tom Loucas, Susie Chandler, David Meldrum, and Andrew Pickles. “Efficacy of Three Screening Instruments in the Identification of Autistic-Spectrum Disorders.” *British Journal of Psychiatry* 191, no. 6 (December 2007): 554–59. https://doi.org/10.1192/bjp.bp.107.040196.

Chaste, Pauline, Lambertus Klei, Stephan J. Sanders, Vanessa Hus, Michael T. Murtha, Jennifer K. Lowe, A. Jeremy Willsey, et al. “A Genome-Wide Association Study of Autism Using the Simons Simplex Collection: Does Reducing Phenotypic Heterogeneity in Autism

Increase Genetic Homogeneity?" *Biological Psychiatry* 77, no. 9 (May 2015): 775–84. https://doi.org/10.1016/j.biopsych.2014.09.017.

Chawarska, Katarzyna, Ami Klin, Rhea Paul, Suzanne Macari, and Fred Volkmar. "A Prospective Study of Toddlers with ASD: Short-Term Diagnostic and Cognitive Outcomes." *Journal of Child Psychology and Psychiatry* 50, no. 10 (October 2009): 1235–45. https://doi.org/10.1111/j.1469-7610.2009.02101.x.

Chen, Jung-Hung, I-Hsin Lin, Chung-Kai Sun, Ling Yang, Thomas Y. Hsueh, Chin-Tsung Ting, and Tung-Hu Tsai. "Transplacental Transfer of Acetaminophen in Pregnant Rats." *Biomedicine & Pharmacotherapy* 154 (October 2022): 113613. https://doi.org/10.1016/j.biopha.2022.113613.

Chen, Mu-Hong, Tai-Long Pan, Pei-Wen Wang, Ju-Wei Hsu, Kai-Lin Huang, Tung-Ping Su, Cheng-Ta Li, et al. "Prenatal Exposure to Acetaminophen and the Risk of Attention-Deficit/Hyperactivity Disorder: A Nationwide Study in Taiwan." *J Clin Psychiatry*, 2019, 7.

Chen, Qi, Catharina A. Hartman, Jan Haavik, Jaanus Harro, Kari Klungsøyr, Tor-Arne Hegvik, Rob Wanders, et al. "Common Psychiatric and Metabolic Comorbidity of Adult Attention-Deficit/Hyperactivity Disorder: A Population-Based Cross-Sectional Study." *PLoS ONE* 13, no. 9 (September 26, 2018): e0204516. https://doi.org/10.1371/journal.pone.0204516.

Chen, Robert Yuzen, Jordan Robert Feltes, William Shun Tzeng, Zoe Yunzhu Lu, Michael Pan, Nan Zhao, Rebecca Talkin, Kavon Javaherian, Anne Glowinski, and Will Ross. "Phone-Based Interventions in Adolescent Psychiatry: A Perspective and Proof of Concept Pilot Study With a Focus on Depression and Autism." *JMIR Research Protocols* 6, no. 6 (June 16, 2017): e114. https://doi.org/10.2196/resprot.7245.

Chen, Shao-wei, Xue-shan Zhong, Li-na Jiang, Xue-yan Zheng, Yi-quan Xiong, Shu-juan Ma, Min Qiu, Shu-ting Huo, Jing Ge, and Qing Chen. "Maternal Autoimmune Diseases and the Risk of Autism Spectrum Disorders in Offspring: A Systematic Review and Meta-Analysis." *Behavioural Brain Research* 296 (January 2016): 61–69. https://doi.org/10.1016/j.bbr.2015.08.035.

Chen, Yu, Jiacheng Dai, Longfei Tang, Tatiana Mikhailova, Qiuman Liang, Miao Li, Jiaqi Zhou, et al. "Neuroimmune Transcriptome Changes in Patient Brains of Psychiatric and Neurological Disorders." *Molecular Psychiatry* 28, no. 2 (February 2023): 710–21. https://doi.org/10.1038/s41380-022-01854-7.

Chen, Zuyue, Hong Wei, Antti Pertovaara, Jianhong Wang, and Synnöve Carlson. "Anxiety- and Activity-Related Effects of Paracetamol on Healthy and Neuropathic Rats." *Pharmacology Research & Perspectives* 6, no. 1 (February 2018): e00367. https://doi.org/10.1002/prp2.367.

Cheng, Chia-Hsiung, Pei-Ying S. Chan, Yu-Wei Hsieh, and Kuan-Fu Chen. “A Meta-Analysis of Mismatch Negativity in Children with Attention Deficit-Hyperactivity Disorders.” *Neuroscience Letters* 612 (January 26, 2016): 132–37. https://doi.org/10.1016/j.neulet.2015.11.033.

Child Mind Institute. “What Should an Evaluation for Autism Look Like?” Accessed May 14, 2023. https://childmind.org/article/what-should-evaluation-autism-look-like/.

Choi, Jae Eun, Felicia Widjaja, Milo Careaga, Stephen Bent, Paul Ashwood, and Robert L. Hendren. “Change in Plasma Cytokine Levels During Risperidone Treatment in Children with Autism.” *Journal of Child and Adolescent Psychopharmacology* 24, no. 10 (December 2014): 586–89. https://doi.org/10.1089/cap.2013.0108.

Chou, I.-Ching, Yu-Tzu Chang, Zheng-Nan Chin, Chih-Hsin Muo, Fung-Chang Sung, Huang-Tsung Kuo, Chang-Hai Tsai, and Chia-Hung Kao. “Correlation between Epilepsy and Attention Deficit Hyperactivity Disorder: A Population-Based Cohort Study.” *PloS One* 8, no. 3 (2013): e57926. https://doi.org/10.1371/journal.pone.0057926.

Christakis, Dimitri A. “Rethinking Attention-Deficit/Hyperactivity Disorder.” *JAMA Pediatrics* 170, no. 2 (February 1, 2016): 109–10. https://doi.org/10.1001/jamapediatrics.2015.3372.

Christensen, Jakob, Therese Koops Grønborg, Merete Juul Sørensen, Diana Schendel, Erik Thorlund Parner, Lars Henning Pedersen, and Mogens Vestergaard. “Prenatal Valproate Exposure and Risk of Autism Spectrum Disorders and Childhood Autism.” *JAMA* 309, no. 16 (April 24, 2013): 1696. https://doi.org/10.1001/jama.2013.2270.

Christensen, Jakob, Lars Pedersen, Yuelian Sun, Julie Werenberg Dreier, Isabell Brikell, and Søren Dalsgaard. “Association of Prenatal Exposure to Valproate and Other Antiepileptic Drugs With Risk for Attention-Deficit/Hyperactivity Disorder in Offspring.” *JAMA Network Open* 2, no. 1 (January 4, 2019): e186606. https://doi.org/10.1001/jamanetworkopen.2018.6606.

Chronis-Tuscano, Andrea, Brooke S. G. Molina, William E. Pelham, Brooks Applegate, Allison Dahlke, Meghan Overmyer, and Benjamin B. Lahey. “Very Early Predictors of Adolescent Depression and Suicide Attempts in Children with Attention-Deficit/Hyperactivity Disorder.” *Archives of General Psychiatry* 67, no. 10 (October 2010): 1044–51. https://doi.org/10.1001/archgenpsychiatry.2010.127.

Clark, T, C Feehan, Raddlebarn Road, and Selly Oak. “Autistic Symptoms in Children with Attention De®cit-Hyperactivity Disorder.” *Adolescent Psychiatry* 8, no. 1 (1999).

Cohrs, Austin C., and Douglas L. Leslie. “Depression in Parents of Children Diagnosed with Autism Spectrum Disorder: A Claims-Based Analysis.” *Journal of Autism and Developmental Disorders* 47, no. 5 (May 2017): 1416–22. https://doi.org/10.1007/s10803-017-3063-y.

Comi, Anne M., Andrew W. Zimmerman, Virginia H. Frye, Paul A. Law, and Joseph N. Peeden. “Familial Clustering of Autoimmune Disorders and Evaluation of Medical Risk Factors in Autism.” *Journal of Child Neurology* 14, no. 6 (June 1999): 388–94. https://doi.org/10.1177/088307389901400608.

"Conners 3rd Edition." Accessed May 30, 2023. https://www.pearsonassessments.com/store/usassessments/en/Store/Professional-Assessments/Behavior/Comprehensive/Conners-3rd-Edition/p/100000523.html.

Conners, C Keith, Jeffery N Epstein, Adrian Angold, and John Klaric. "Continuous Performance Test Performance in a Normative Epidemiological Sample," 2003.

Coolican, Jamesie, Susan E. Bryson, and Lonnie Zwaigenbaum. "Brief Report: Data on the Stanford–Binet Intelligence Scales (5th Ed.) in Children with Autism Spectrum Disorder." *Journal of Autism and Developmental Disorders* 38, no. 1 (January 2008): 190–97. https://doi.org/10.1007/s10803-007-0368-2.

Cooper, Miriam, Joanna Martin, Kate Langley, Marian Hamshere, and Anita Thapar. "Autistic Traits in Children with ADHD Index Clinical and Cognitive Problems." *European Child & Adolescent Psychiatry* 23, no. 1 (January 2014): 23–34. https://doi.org/10.1007/s00787-013-0398-6.

Cortese, Samuele, Shihua Sun, Junhua Zhang, Esha Sharma, Zheng Chang, Ralf Kuja-Halkola, Catarina Almqvist, Henrik Larsson, and Stephen V. Faraone. "Association between Attention Deficit Hyperactivity Disorder and Asthma: A Systematic Review and Meta-Analysis and a Swedish Population-Based Study." *The Lancet. Psychiatry* 5, no. 9 (September 2018): 717–26. https://doi.org/10.1016/S2215-0366(18)30224-4.

Costello, Anna B., and Jason Osborne. "Best Practices in Exploratory Factor Analysis: Four Recommendations for Getting the Most from Your Analysis." Accessed May 15, 2023. https://doi.org/10.7275/JYJ1-4868.

Cree, Robyn A., Rebecca H. Bitsko, Melissa L. Danielson, Valentine Wanga, Joseph Holbrook, Kate Flory, Lorraine F. Kubicek, Steven W. Evans, Julie Sarno Owens, and Steven P. Cuffe. "Surveillance of ADHD Among Children in the United States: Validity and Reliability of Parent Report of Provider Diagnosis." *Journal of Attention Disorders* 27, no. 2 (January 1, 2023): 111–23. https://doi.org/10.1177/10870547221131979.

Crippa, J. A. S., J. E. C. Hallak, J. P. Machado-de-Sousa, R. H. C. Queiroz, M. Bergamaschi, M. H. N. Chagas, and A. W. Zuardi. "Cannabidiol for the Treatment of *Cannabis* Withdrawal Syndrome: A Case Report: CBD and Cannabis Withdrawal." *Journal of Clinical Pharmacy and Therapeutics* 38, no. 2 (April 2013): 162–64. https://doi.org/10.1111/jcpt.12018.

Critchfield, J. William, Saskia Van Hemert, Michael Ash, Linda Mulder, and Paul Ashwood. "The Potential Role of Probiotics in the Management of Childhood Autism Spectrum Disorders." *Gastroenterology Research and Practice* 2011 (2011): 1–8. https://doi.org/10.1155/2011/161358.

Croen, Lisa A., Daniel V. Najjar, Bruce Fireman, and Judith K. Grether. "Maternal and Paternal Age and Risk of Autism Spectrum Disorders." *Archives of Pediatrics & Adolescent Medicine* 161, no. 4 (April 1, 2007): 334–40. https://doi.org/10.1001/archpedi.161.4.334.

Culpin, Iryna, Becky Mars, Rebecca M. Pearson, Jean Golding, Jon Heron, Isidora Bubak, Peter Carpenter, Cecilia Magnusson, David Gunnell, and Dheeraj Rai. "Autistic Traits and Suicidal Thoughts, Plans, and Self-Harm in Late Adolescence: Population-Based Cohort

Study." *Journal of the American Academy of Child & Adolescent Psychiatry* 57, no. 5 (May 2018): 313-320.e6. https://doi.org/10.1016/j.jaac.2018.01.023.

Cummings, Janet R., Frances L. Lynch, Kristal C. Rust, Karen J. Coleman, Jeanne M. Madden, Ashli A. Owen-Smith, Vincent M. Yau, et al. "Health Services Utilization Among Children With and Without Autism Spectrum Disorders." *Journal of Autism and Developmental Disorders* 46, no. 3 (March 2016): 910–20. https://doi.org/10.1007/s10803-015-2634-z.

Curran, Eileen A., Christina Dalman, Patricia M. Kearney, Louise C. Kenny, John F. Cryan, Timothy G. Dinan, and Ali S. Khashan. "Association Between Obstetric Mode of Delivery and Autism Spectrum Disorder: A Population-Based Sibling Design Study." *JAMA Psychiatry* 72, no. 9 (September 2015): 935–42. https://doi.org/10.1001/jamapsychiatry.2015.0846.

Curran, Laura K., Craig J. Newschaffer, Li-Ching Lee, Stephen O. Crawford, Michael V. Johnston, and Andrew W. Zimmerman. "Behaviors Associated With Fever in Children With Autism Spectrum Disorders." *Pediatrics* 120, no. 6 (December 1, 2007): e1386–92. https://doi.org/10.1542/peds.2007-0360.

Damkier, Per. "Pledging My Time: In Utero Exposure to Acetaminophen and Childhood Neurodevelopment." *Paediatric and Perinatal Epidemiology*, May 3, 2023. https://doi.org/10.1111/ppe.12985.

———. "Simple Twist of Fate: In Utero Exposure to Acetaminophen and Risk of Childhood Attention Deficit Hyperactivity Disorder." *Paediatric and Perinatal Epidemiology* 34, no. 3 (May 2020): 230–32. https://doi.org/10.1111/ppe.12654.

Damkier, Per, Brian Cleary, Corinna Weber-Schoendorfer, Svetlana Shechtman, Matteo Cassina, Alice Panchaud, and Orna Diav-Cirtin. "Handle with Care—Interpretation, Synthesis and Dissemination of Data on Paracetamol in Pregnancy." *Nature Reviews Endocrinology* 18, no. 3 (March 2022): 191–191. https://doi.org/10.1038/s41574-021-00605-y.

Damkier, Per, Anthony R. Scialli, and Shari I. Lusskin. "Acetaminophen in Pregnancy and Adverse Childhood Neurodevelopment." *JAMA Pediatrics* 171, no. 4 (April 1, 2017): 396. https://doi.org/10.1001/jamapediatrics.2016.5049.

Danielson, Melissa L., Rebecca H. Bitsko, Reem M. Ghandour, Joseph R. Holbrook, Michael D. Kogan, and Stephen J. Blumberg. "Prevalence of Parent-Reported ADHD Diagnosis and Associated Treatment Among U.S. Children and Adolescents, 2016." *Journal of Clinical Child and Adolescent Psychology : The Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53* 47, no. 2 (2018): 199–212. https://doi.org/10.1080/15374416.2017.1417860.

Darwish, Amira Hamed, Tarek Mohamed Elgohary, and Nahla A. Nosair. "Serum Interleukin-6 Level in Children With Attention-Deficit Hyperactivity Disorder (ADHD)." *Journal of Child Neurology* 34, no. 2 (February 1, 2019): 61–67. https://doi.org/10.1177/0883073818809831.

Davies, M. H., J. M. Ngong, M. Yucesoy, S. K. Acharya, C. O. Mills, J. B. Weaver, R. H. Waring, and E. Elias. "The Adverse Influence of Pregnancy upon Sulphation: A Clue to the Pathogenesis of Intrahepatic Cholestasis of Pregnancy?" *Journal of Hepatology* 21, no. 6 (December 1994): 1127–34. https://doi.org/10.1016/s0168-8278(05)80630-0.

DaWalt, Leann Smith, Julie Lounds Taylor, Somer Bishop, Laura J. Hall, Jessica Dykstra Steinbrenner, Bonnie Kraemer, Kara A. Hume, and Samuel L. Odom. "Sex Differences in Social Participation of High School Students with Autism Spectrum Disorder." *Autism Research* 13, no. 12 (December 2020): 2155–63. https://doi.org/10.1002/aur.2348.

"DAWBA | Youthinmind." Accessed May 28, 2023. https://youthinmind.com/products-and-services/dawba/.

De Petrocellis, Luciano, Alessia Ligresti, Aniello Schiano Moriello, Marco Allarà, Tiziana Bisogno, Stefania Petrosino, Colin G Stott, and Vincenzo Di Marzo. "Effects of Cannabinoids and Cannabinoid-Enriched *Cannabis* Extracts on TRP Channels and Endocannabinoid Metabolic Enzymes: Novel Pharmacology of Minor Plant Cannabinoids." *British Journal of Pharmacology* 163, no. 7 (August 2011): 1479–94. https://doi.org/10.1111/j.1476-5381.2010.01166.x.

Dean, Shannon L., Jessica F. Knutson, Desiree L. Krebs-Kraft, and Margaret M. McCarthy. "Prostaglandin E2 Is an Endogenous Modulator of Cerebellar Development and Complex Behavior during a Sensitive Postnatal Period." *The European Journal of Neuroscience* 35, no. 8 (April 2012): 1218–29. https://doi.org/10.1111/j.1460-9568.2012.08032.x.

Deavenport-Saman, Alexis, Yang Lu, Kathryn Smith, and Larry Yin. "Do Children with Autism Overutilize the Emergency Department? Examining Visit Urgency and Subsequent Hospital Admissions." *Maternal and Child Health Journal* 20, no. 2 (February 2016): 306–14. https://doi.org/10.1007/s10995-015-1830-y.

Deiana, Serena, Akihito Watanabe, Yuki Yamasaki, Naoki Amada, Marlene Arthur, Shona Fleming, Hilary Woodcock, et al. "Plasma and Brain Pharmacokinetic Profile of Cannabidiol (CBD), Cannabidivarine (CBDV), Δ9-Tetrahydrocannabivarin (THCV) and Cannabigerol (CBG) in Rats and Mice Following Oral and Intraperitoneal Administration and CBD Action on Obsessive–Compulsive Behaviour." *Psychopharmacology* 219, no. 3 (February 2012): 859–73. https://doi.org/10.1007/s00213-011-2415-0.

Del Valle Rubido, Marta, Eric Hollander, James T. McCracken, Frederick Shic, Jana Noeldeke, Lauren Boak, Omar Khwaja, Shamil Sadikhov, Paulo Fontoura, and Daniel Umbricht. "Exploring Social Biomarkers in High-Functioning Adults with Autism and Asperger's Versus Healthy Controls: A Cross-Sectional Analysis." *Journal of Autism and Developmental Disorders* 50, no. 12 (December 2020): 4412–30. https://doi.org/10.1007/s10803-020-04493-5.

Del Valle Rubido, Marta, James T. McCracken, Eric Hollander, Frederick Shic, Jana Noeldeke, Lauren Boak, Omar Khwaja, Shamil Sadikhov, Paulo Fontoura, and Daniel Umbricht. "In Search of Biomarkers for Autism Spectrum Disorder: Exploratory Biomarkers in Autism." *Autism Research* 11, no. 11 (November 2018): 1567–79. https://doi.org/10.1002/aur.2026.

DelGiudice-Asch, Gina, Lorraine Simon, James Schmeidler, Charlotte Cunningham-Rundles, and Eric Hollander. "Brief Report: A Pilot Open Clinical Trial of Intravenous Immunoglobulin in Childhood Autism," n.d.

Dell'Osso, L., B. Carpita, C. Gesi, I.M. Cremone, M. Corsi, E. Massimetti, D. Muti, et al. "Subthreshold Autism Spectrum Disorder in Patients with Eating Disorders." *Comprehensive Psychiatry* 81 (February 2018): 66–72. https://doi.org/10.1016/j.comppsych.2017.11.007.

Dell'Osso, Liliana, Ivan Mirko Cremone, Giulia Amatori, Andrea Cappelli, Alessandro Cuomo, Stefano Barlati, Gabriele Massimetti, et al. "Investigating the Relationship between Autistic Traits, Ruminative Thinking, and Suicidality in a Clinical Sample of Subjects with Bipolar Disorder and Borderline Personality Disorder." *Brain Sciences* 11, no. 5 (May 12, 2021): 621. https://doi.org/10.3390/brainsci11050621.

Demontis, Ditte, Raymond K. Walters, Joanna Martin, Manuel Mattheisen, Thomas D. Als, Esben Agerbo, Gísli Baldursson, et al. "Discovery of the First Genome-Wide Significant Risk Loci for Attention-Deficit/Hyperactivity Disorder." *Nature Genetics* 51, no. 1 (January 2019): 63–75. https://doi.org/10.1038/s41588-018-0269-7.

Derecki, Noël C., James C. Cronk, Zhenjie Lu, Eric Xu, Stephen B. G. Abbott, Patrice G. Guyenet, and Jonathan Kipnis. "Wild-Type Microglia Arrest Pathology in a Mouse Model of Rett Syndrome." *Nature* 484, no. 7392 (April 5, 2012): 105–9. https://doi.org/10.1038/nature10907.

Derecki, Noël C., Natalie Katzmarski, Jonathan Kipnis, and Melanie Meyer-Luehmann. "Microglia as a Critical Player in Both Developmental and Late-Life CNS Pathologies." *Acta Neuropathologica* 128, no. 3 (September 2014): 333–45. https://doi.org/10.1007/s00401-014-1321-z.

Desbonnet, Lieve, Colm Mp O'Tuathaigh, and John L. Waddington. "Modeling Schizophrenia: Uncovering Novel Therapeutic Targets." *Expert Review of Clinical Pharmacology* 5, no. 6 (November 2012): 667–76. https://doi.org/10.1586/ecp.12.57.

Deverman, Benjamin E., and Paul H. Patterson. "Cytokines and CNS Development." *Neuron* 64, no. 1 (October 15, 2009): 61–78. https://doi.org/10.1016/j.neuron.2009.09.002.

Devinsky, Orrin, Maria Roberta Cilio, Helen Cross, Javier Fernandez-Ruiz, Jacqueline French, Charlotte Hill, Russell Katz, et al. "Cannabidiol: Pharmacology and Potential Therapeutic Role in Epilepsy and Other Neuropsychiatric Disorders." *Epilepsia* 55, no. 6 (June 2014): 791–802. https://doi.org/10.1111/epi.12631.

Devlin, Bernie, Nadine Melhem, and Kathryn Roeder. "Do Common Variants Play a Role in Risk for Autism? Evidence and Theoretical Musings." *Brain Research* 1380 (March 2011): 78–84. https://doi.org/10.1016/j.brainres.2010.11.026.

Di Marzo, Vincenzo, Maurizio Bifulco, and Luciano De Petrocellis. "The Endocannabinoid System and Its Therapeutic Exploitation." *Nature Reviews. Drug Discovery* 3, no. 9 (September 2004): 771–84. https://doi.org/10.1038/nrd1495.

DOENNI, V.M., J.M. GRAY, C.M. SONG, S. PATEL, M.N. HILL, and Q.J. PITTMAN.

"Deficient Adolescent Social Behavior Following Early-Life Inflammation Is Ameliorated by Augmentation of Anandamide Signaling." *Brain, Behavior, and Immunity* 58 (November 2016): 237–47. https://doi.org/10.1016/j.bbi.2016.07.152.

Doernberg, Ellen, and Eric Hollander. "Neurodevelopmental Disorders (ASD and ADHD): DSM-5, ICD-10, and ICD-11." *CNS Spectrums* 21, no. 4 (August 2016): 295–99. https://doi.org/10.1017/S1092852916000262.

Doyle-Thomas, Krissy A.R., Emma G. Duerden, Margot J. Taylor, Jason P. Lerch, Latha V. Soorya, A. Ting Wang, Jin Fan, Eric Hollander, and Evdokia Anagnostou. "Effects of Age and Symptomatology on Cortical Thickness in Autism Spectrum Disorders." *Research in Autism Spectrum Disorders* 7, no. 1 (January 2013): 141–50. https://doi.org/10.1016/j.rasd.2012.08.004.

Drechsler, Renate, Silvia Brem, Daniel Brandeis, Edna Grünblatt, Gregor Berger, and Susanne Walitza. "ADHD: Current Concepts and Treatments in Children and Adolescents." *Neuropediatrics* 51, no. 5 (October 2020): 315–35. https://doi.org/10.1055/s-0040-1701658.

Dreyer, Bernard. "AAP Statement on U.S. Preventive Services Task Force Final Recommendation Statement on Autism Screening," February 16, 2016. https://www.superdoctors.com/article/AAP-Statement-on-US-Preventive-Services-Task-Force-Final-Recommendation-Statement-on-Autism-Screening/39e722b4-b152-4f61-9a4a-89231be50252.html.

Drtilkova, Ivana, Omar Sery, Pavel Theiner, Alena Uhrova, Marketa Zackova, Blanka Balastikova, and Vladimir Znojil. "Clinical and Molecular-Genetic Markers of ADHD in Children." *Neuro Endocrinology Letters* 29, no. 3 (June 2008): 320–27.

Dudas, Robert B., Chris Lovejoy, Sarah Cassidy, Carrie Allison, Paula Smith, and Simon Baron-Cohen. "The Overlap between Autistic Spectrum Conditions and Borderline Personality Disorder." Edited by Michel Botbol. *PLOS ONE* 12, no. 9 (September 8, 2017): e0184447. https://doi.org/10.1371/journal.pone.0184447.

Dunn, Geoffrey A., Joel T. Nigg, and Elinor L. Sullivan. "Neuroinflammation as a Risk Factor for Attention Deficit Hyperactivity Disorder." *Pharmacology, Biochemistry, and Behavior* 182 (July 2019): 22–34. https://doi.org/10.1016/j.pbb.2019.05.005.

Durkin, Maureen S., Matthew J. Maenner, Craig J. Newschaffer, Li-Ching Lee, Christopher M. Cunniff, Julie L. Daniels, Russell S. Kirby, et al. "Advanced Parental Age and the Risk of Autism Spectrum Disorder." *American Journal of Epidemiology* 168, no. 11 (December 1, 2008): 1268–76. https://doi.org/10.1093/aje/kwn250.

Dworzynski, Katharina, Francesca Happé, Patrick Bolton, and Angelica Ronald. "Relationship Between Symptom Domains in Autism Spectrum Disorders: A Population Based Twin Study." *Journal of Autism and Developmental Disorders* 39, no. 8 (August 2009): 1197–1210. https://doi.org/10.1007/s10803-009-0736-1.

Eaton, Christopher, Kayley Roarty, Nimisha Doval, Smitarani Shetty, Karen Goodall, and Sinead M. Rhodes. "The Prevalence of Attention Deficit/Hyperactivity Disorder Symptoms in Children and Adolescents With Autism Spectrum Disorder Without

Intellectual Disability: A Systematic Review." *Journal of Attention Disorders*, June 7, 2023, 108705472311774. https://doi.org/10.1177/10870547231177466.

Eisenberg, Ian W., Gregory L. Wallace, Lauren Kenworthy, Stephen J. Gotts, and Alex Martin. "Insistence on Sameness Relates to Increased Covariance of Gray Matter Structure in Autism Spectrum Disorder." *Molecular Autism* 6, no. 1 (December 2015): 54. https://doi.org/10.1186/s13229-015-0047-7.

Elberling, Hanne, Allan Linneberg, Else Marie Olsen, Tine Houmann, Charlotte Ulrikka Rask, Robert Goodman, and Anne Mette Skovgaard. "Infancy Predictors of Hyperkinetic and Pervasive Developmental Disorders at Ages 5-7 Years: Results from the Copenhagen Child Cohort CCC2000." *Journal of Child Psychology and Psychiatry* 55, no. 12 (December 2014): 1328–35. https://doi.org/10.1111/jcpp.12256.

Elison, Jed T., Jason J. Wolff, J. Steven Reznick, Kelly N. Botteron, Annette M. Estes, Hongbin Gu, Heather C. Hazlett, et al. "Repetitive Behavior in 12-Month-Olds Later Classified With Autism Spectrum Disorder." *Journal of the American Academy of Child and Adolescent Psychiatry* 53, no. 11 (November 2014): 1216–24. https://doi.org/10.1016/j.jaac.2014.08.004.

Epstein, Jeffery N, Alaatin Erkanli, C Keith Conners, John Klaric, Jane E Costello, and Adrian Angold. "Relations Between Continuous Performance Test Performance Measures and ADHD Behaviors." *Journal of Abnormal Child Psychology*, 2003.

Ernst, Andreas, Nis Brix, Lea L. B. Lauridsen, Jørn Olsen, Erik T. Parner, Zeyan Liew, Lars H. Olsen, and Cecilia H. Ramlau-Hansen. "Acetaminophen (Paracetamol) Exposure During Pregnancy and Pubertal Development in Boys and Girls From a Nationwide Puberty Cohort." *American Journal of Epidemiology* 188, no. 1 (January 1, 2019): 34–46. https://doi.org/10.1093/aje/kwy193.

Eslamimehr, Shakiba, A. Daniel Jones, Thilani M. Anthony, S. Hasan Arshad, John W. Holloway, Susan Ewart, Rui Luo, et al. "Association of Prenatal Acetaminophen Use and Acetaminophen Metabolites with DNA Methylation of Newborns: Analysis of Two Consecutive Generations of the Isle of Wight Birth Cohort," February 2, 2022.

Estes, Myka L., and A. Kimberley McAllister. "Immune Mediators in the Brain and Peripheral Tissues in Autism Spectrum Disorder." *Nature Reviews. Neuroscience* 16, no. 8 (August 2015): 469–86. https://doi.org/10.1038/nrn3978.

———. "Immune Mediators in the Brain and Peripheral Tissues in Autism Spectrum Disorder." *Nature Reviews. Neuroscience* 16, no. 8 (August 2015): 469–86. https://doi.org/10.1038/nrn3978.

———. "Maternal Immune Activation: Implications for Neuropsychiatric Disorders." *Science (New York, N.Y.)* 353, no. 6301 (August 19, 2016): 772–77. https://doi.org/10.1126/science.aag3194.

Evans, David W, James F Leckman, Alice Carter, J Steven Reznick, Desiree Henshaw, Robert A King, and David Pauls. "Ritual, Habit, and Perfectionism: The Prevalence and

Development of Compulsive-Like Behavior in Normal Young Children," n.d.

Evans, David W., Mirko Uljarević, Laina G. Lusk, Eva Loth, and Thomas Frazier. "Development of Two Dimensional Measures of Restricted and Repetitive Behavior in Parents and Children." *Journal of the American Academy of Child & Adolescent Psychiatry* 56, no. 1 (January 2017): 51–58. https://doi.org/10.1016/j.jaac.2016.10.014.

Evers, M, C Cunningham-Rundles, and E Hollander. "Heat Shock Protein 90 Antibodies in Autism." *Molecular Psychiatry* 7, no. S2 (August 1, 2002): S26–28. https://doi.org/10.1038/sj.mp.4001171.

"Eye Vergence Responses During an Attention Task in Adults With ADHD and Clinical Controls." Accessed June 9, 2023. https://doi.org/10.1177/1087054719897806.

Factor, Reina S., Emma E. Condy, Julee P. Farley, and Angela Scarpa. "Brief Report: Insistence on Sameness, Anxiety, and Social Motivation in Children with Autism Spectrum Disorder." *Journal of Autism and Developmental Disorders* 46, no. 7 (July 2016): 2548–54. https://doi.org/10.1007/s10803-016-2781-x.

Fan, Jin, Silvia Bernardi, Nicholas T. Dam, Evdokia Anagnostou, Xiaosi Gu, Laura Martin, Yunsoo Park, et al. "Functional Deficits of the Attentional Networks in Autism." *Brain and Behavior* 2, no. 5 (September 2012): 647–60. https://doi.org/10.1002/brb3.90.

Faraone, Stephen V., Tobias Banaschewski, David Coghill, Yi Zheng, Joseph Biederman, Mark A. Bellgrove, Jeffrey H. Newcorn, et al. "The World Federation of ADHD International Consensus Statement: 208 Evidence-Based Conclusions about the Disorder." *Neuroscience and Biobehavioral Reviews* 128 (September 2021): 789–818. https://doi.org/10.1016/j.neubiorev.2021.01.022.

Faraone, Stephen V., and Henrik Larsson. "Genetics of Attention Deficit Hyperactivity Disorder." *Molecular Psychiatry* 24, no. 4 (2019): 562–75. https://doi.org/10.1038/s41380-018-0070-0.

Fedak, Kristen M., Autumn Bernal, Zachary A. Capshaw, and Sherilyn Gross. "Applying the Bradford Hill Criteria in the 21st Century: How Data Integration Has Changed Causal Inference in Molecular Epidemiology." *Emerging Themes in Epidemiology* 12, no. 1 (September 30, 2015): 14. https://doi.org/10.1186/s12982-015-0037-4.

Feldman, Inbal, Judah Koller, Eli R. Lebowitz, Cory Shulman, Esther Ben Itzchak, and Ditza A. Zachor. "Family Accommodation in Autism Spectrum Disorder." *Journal of Autism and Developmental Disorders* 49, no. 9 (September 2019): 3602–10. https://doi.org/10.1007/s10803-019-04078-x.

Feliciano, Pamela, Xueya Zhou, Irina Astrovskaya, Tychele N. Turner, Tianyun Wang, Leo Brueggeman, Rebecca Barnard, et al. "Exome Sequencing of 457 Autism Families Recruited Online Provides Evidence for Autism Risk Genes." *Npj Genomic Medicine* 4, no. 1 (August 23, 2019): 19. https://doi.org/10.1038/s41525-019-0093-8.

Ferguson, Bradley J., Sarah Marler, Lily L. Altstein, Evon Batey Lee, Jill Akers, Kristin Sohl, Aaron McLaughlin, et al. "Psychophysiological Associations with Gastrointestinal Symptomatology in Autism Spectrum Disorder: ASD Psychophysiology &

Gastrointestinal Issues." *Autism Research* 10, no. 2 (February 2017): 276–88. https://doi.org/10.1002/aur.1646.

Finegold, Sydney M. "State of the Art; Microbiology in Health and Disease. Intestinal Bacterial Flora in Autism." *Anaerobe* 17, no. 6 (December 2011): 367–68. https://doi.org/10.1016/j.anaerobe.2011.03.007.

Finegold, Sydney M., Scot E. Dowd, Viktoria Gontcharova, Chengxu Liu, Kathleen E. Henley, Randall D. Wolcott, Eunseog Youn, et al. "Pyrosequencing Study of Fecal Microflora of Autistic and Control Children." *Anaerobe* 16, no. 4 (August 2010): 444–53. https://doi.org/10.1016/j.anaerobe.2010.06.008.

Fitzpatrick, Sarah, Craig Erickson, Laura Srivorakiat, Logan Wink, and Ernest Pedapati. "Aggression in Autism Spectrum Disorder: Presentation and Treatment Options." *Neuropsychiatric Disease and Treatment*, June 2016, 1525. https://doi.org/10.2147/NDT.S84585.

Flores, Cindi G., Gregory Valcante, Steve Guter, Annette Zaytoun, Emily Wray, Lindsay Bell, Suma Jacob, et al. "Repetitive Behavior Profiles: Consistency across Autism Spectrum Disorder Cohorts and Divergence from Prader–Willi Syndrome." *Journal of Neurodevelopmental Disorders* 3, no. 4 (December 2011): 316–24. https://doi.org/10.1007/s11689-011-9094-3.

Foldi, Claire J., Darryl W. Eyles, John J. McGrath, and Thomas H. J. Burne. "Advanced Paternal Age Is Associated with Alterations in Discrete Behavioural Domains and Cortical Neuroanatomy of C57BL/6J Mice." *The European Journal of Neuroscience* 31, no. 3 (February 2010): 556–64. https://doi.org/10.1111/j.1460-9568.2010.07074.x.

Földy, Csaba, Robert C. Malenka, and Thomas C. Südhof. "Autism-Associated Neuroligin-3 Mutations Commonly Disrupt Tonic Endocannabinoid Signaling." *Neuron* 78, no. 3 (May 8, 2013): 498–509. https://doi.org/10.1016/j.neuron.2013.02.036.

For the Autism and Developmental Disorders Inpatient Research Collaborative (ADDIRC), Matthew Siegel, Kahsi A. Smith, Carla Mazefsky, Robin L. Gabriels, Craig Erickson, Desmond Kaplan, Eric M. Morrow, Logan Wink, and Susan L. Santangelo. "The Autism Inpatient Collection: Methods and Preliminary Sample Description." *Molecular Autism* 6, no. 1 (December 2015): 61. https://doi.org/10.1186/s13229-015-0054-8.

Foreman, David, Stephanie Morton, and Tamsin Ford. "Exploring the Clinical Utility of the Development And Well-Being Assessment (DAWBA) in the Detection of Hyperkinetic Disorders and Associated Diagnoses in Clinical Practice." *Journal of Child Psychology and Psychiatry* 50, no. 4 (2009): 460–70. https://doi.org/10.1111/j.1469-7610.2008.02017.x.

Fox, Elizabeth, David Amaral, and Judy Van De Water. "Maternal and Fetal Antibrain Antibodies in Development and Disease." *Developmental Neurobiology* 72, no. 10 (October 2012): 1327–34. https://doi.org/10.1002/dneu.22052.

Frazier, Thomas W., Eric A. Youngstrom, Cynthia S. Kubu, Leslie Sinclair, and Ali Rezai. "Exploratory and Confirmatory Factor Analysis of the Autism Diagnostic Interview-Revised." *Journal of Autism and Developmental Disorders* 38, no. 3 (March 2008): 474–

80. https://doi.org/10.1007/s10803-007-0415-z.

Freitag, Christine M., Susann Hänig, Anna Schneider, Christiane Seitz, Haukur Palmason, Wolfgang Retz, and Jobst Meyer. "Biological and Psychosocial Environmental Risk Factors Influence Symptom Severity and Psychiatric Comorbidity in Children with ADHD." *Journal of Neural Transmission (Vienna, Austria: 1996)* 119, no. 1 (January 2012): 81–94. https://doi.org/10.1007/s00702-011-0659-9.

Frick, Matilda A., Karin C. Brocki, Linda Halldner Henriksson, and Johan Lundin Kleberg. "Disrupted Attention to Other's Eyes Is Linked to Symptoms of ADHD in Childhood." *Child Psychiatry & Human Development*, January 17, 2022. https://doi.org/10.1007/s10578-022-01316-9.

Frye, C. A., E. Bo, G. Calamandrei, L. Calzà, F. Dessì-Fulgheri, M. Fernández, L. Fusani, et al. "Endocrine Disrupters: A Review of Some Sources, Effects, and Mechanisms of Actions on Behaviour and Neuroendocrine Systems." *Journal of Neuroendocrinology* 24, no. 1 (January 2012): 144–59. https://doi.org/10.1111/j.1365-2826.2011.02229.x.

Frye, Richard E. "Mitochondrial Dysfunction in Autism Spectrum Disorder: Unique Abnormalities and Targeted Treatments." *Seminars in Pediatric Neurology* 35 (October 2020): 100829. https://doi.org/10.1016/j.spen.2020.100829.

Frye, Richard E., Janet Cakir, Shannon Rose, Raymond F. Palmer, Christine Austin, Paul Curtin, and Manish Arora. "Mitochondria May Mediate Prenatal Environmental Influences in Autism Spectrum Disorder." *Journal of Personalized Medicine* 11, no. 3 (March 18, 2021): 218. https://doi.org/10.3390/jpm11030218.

Frye, Richard E, and S Jill James. "Metabolic Pathology of Autism in Relation to Redox Metabolism." *Biomarkers in Medicine* 8, no. 3 (March 2014): 321–30. https://doi.org/10.2217/bmm.13.158.

Fulceri, Francesca, Antonio Narzisi, Fabio Apicella, Giulia Balboni, Sara Baldini, Jenny Brocchini, Ilaria Domenici, et al. "Application of the Repetitive Behavior Scale-Revised – Italian Version – in Preschoolers with Autism Spectrum Disorder." *Research in Developmental Disabilities* 48 (January 2016): 43–52. https://doi.org/10.1016/j.ridd.2015.10.015.

"Functional Connectivity of Attention-Related Networks in Drug-Naïve Children With ADHD." Accessed June 9, 2023. https://doi.org/10.1177/1087054718802017.

Fung, Lawrence K., Rajneesh Mahajan, Alixandra Nozzolillo, Pilar Bernal, Aaron Krasner, Booil Jo, Daniel Coury, Agnes Whitaker, Jeremy Veenstra-Vanderweele, and Antonio Y. Hardan. "Pharmacologic Treatment of Severe Irritability and Problem Behaviors in Autism: A Systematic Review and Meta-Analysis." *Pediatrics* 137, no. Supplement_2 (February 1, 2016): S124–35. https://doi.org/10.1542/peds.2015-2851K.

Fusaroli, Riccardo, Anna Lambrechts, Dan Bang, Dermot M. Bowler, and Sebastian B. Gaigg. "'Is Voice a Marker for Autism Spectrum Disorder? A Systematic Review and Meta-Analysis': Vocal Production in ASD." *Autism Research* 10, no. 3 (March 2017): 384–407. https://doi.org/10.1002/aur.1678.

G Aman, Michael. “Aberrant Behavior Checklist: Current Identity and Future Developments.” *Clinical and Experimental Pharmacology* 02, no. 03 (2012). https://doi.org/10.4172/2161-1459.1000e114.

Gadow, Kenneth D., and Heather D. Garman. “Social Anhedonia in Children and Adolescents with Autism Spectrum Disorder and Psychiatry Referrals.” *Journal of Clinical Child & Adolescent Psychology* 49, no. 2 (March 3, 2020): 239–50. https://doi.org/10.1080/15374416.2018.1514611.

Garay, Paula A., Elaine Y. Hsiao, Paul H. Patterson, and A.K. McAllister. “Maternal Immune Activation Causes Age- and Region-Specific Changes in Brain Cytokines in Offspring throughout Development.” *Brain, Behavior, and Immunity* 31 (July 2013): 54–68. https://doi.org/10.1016/j.bbi.2012.07.008.

Garbett, K A, E Y Hsiao, S Kálmán, P H Patterson, and K Mirnics. “Effects of Maternal Immune Activation on Gene Expression Patterns in the Fetal Brain.” *Translational Psychiatry* 2, no. 4 (April 3, 2012): e98–e98. https://doi.org/10.1038/tp.2012.24.

Garcia, Kristine L.P., Guanhua Yu, Chiara Nicolini, Bernadeta Michalski, Diego J. Garzon, Victor S. Chiu, Enrico Tongiorgi, Peter Szatmari, and Margaret Fahnestock. “Altered Balance of Proteolytic Isoforms of Pro-Brain-Derived Neurotrophic Factor in Autism.” *Journal of Neuropathology & Experimental Neurology* 71, no. 4 (April 1, 2012): 289–97. https://doi.org/10.1097/NEN.0b013e31824b27e4.

Garcia-Oscos, Francisco, David Peña, Mohammad Housini, Derek Cheng, Diego Lopez, Michael S. Borland, Roberto Salgado-Delgado, et al. “Vagal Nerve Stimulation Blocks Interleukin 6-Dependent Synaptic Hyperexcitability Induced by Lipopolysaccharide-Induced Acute Stress in the Rodent Prefrontal Cortex.” *Brain, Behavior, and Immunity* 43 (January 2015): 149–58. https://doi.org/10.1016/j.bbi.2014.07.020.

Gardener, Hannah, Donna Spiegelman, and Stephen L. Buka. “Perinatal and Neonatal Risk Factors for Autism: A Comprehensive Meta-Analysis.” *Pediatrics* 128, no. 2 (August 2011): 344–55. https://doi.org/10.1542/peds.2010-1036.

———. “Prenatal Risk Factors for Autism: Comprehensive Meta-Analysis.” *The British Journal of Psychiatry: The Journal of Mental Science* 195, no. 1 (July 2009): 7–14. https://doi.org/10.1192/bjp.bp.108.051672.

Gardner, Renee M., Brian K. Lee, Cecilia Magnusson, Dheeraj Rai, Thomas Frisell, Håkan Karlsson, Selma Idring, and Christina Dalman. “Maternal Body Mass Index during Early Pregnancy, Gestational Weight Gain, and Risk of Autism Spectrum Disorders: Results from a Swedish Total Population and Discordant Sibling Study.” *International Journal of Epidemiology* 44, no. 3 (June 2015): 870–83. https://doi.org/10.1093/ije/dyv081.

Gaugler, Trent, Lambertus Klei, Stephan J Sanders, Corneliu A Bodea, Arthur P Goldberg, Ann B Lee, Milind Mahajan, et al. “Most Genetic Risk for Autism Resides with Common Variation.” *Nature Genetics* 46, no. 8 (August 2014): 881–85. https://doi.org/10.1038/ng.3039.

“Gender Identity and Sexual Orientation in Autism Spectrum Disorder.” Accessed May 15, 2023. https://doi.org/10.1177/1362361317714587.

Gershon, J., and Jonathan Gershon. "A Meta-Analytic Review of Gender Differences in ADHD." *Journal of Attention Disorders* 5, no. 3 (January 1, 2002): 143–54. https://doi.org/10.1177/108705470200500302.

Gervin, Kristina, Hedvig Nordeng, Eivind Ystrom, Ted Reichborn-Kjennerud, and Robert Lyle. "Long-Term Prenatal Exposure to Paracetamol Is Associated with DNA Methylation Differences in Children Diagnosed with ADHD." *Clinical Epigenetics* 9, no. 1 (December 2017): 77. https://doi.org/10.1186/s13148-017-0376-9.

Gesundheit, Benjamin, Joshua P. Rosenzweig, David Naor, Bernard Lerer, Ditza A. Zachor, Vaclav Procházka, Michal Melamed, et al. "Immunological and Autoimmune Considerations of Autism Spectrum Disorders." *Journal of Autoimmunity* 44 (August 2013): 1–7. https://doi.org/10.1016/j.jaut.2013.05.005.

Getahun, Darios, Steven J. Jacobsen, Michael J. Fassett, Wansu Chen, Kitaw Demissie, and George G. Rhoads. "Recent Trends in Childhood Attention-Deficit/Hyperactivity Disorder." *JAMA Pediatrics* 167, no. 3 (March 1, 2013): 282. https://doi.org/10.1001/2013.jamapediatrics.401.

Ghanem, Carolina I, J Pérez María, José E Manautou, and Aldo D Mottino. "ACETAMINOPHEN; FROM LIVER TO BRAIN: NEW INSIGHTS INTO DRUG PHARMACOLOGICAL ACTION AND TOXICITY." *Pharmacological Research* 109 (July 2016): 119–31. https://doi.org/10.1016/j.phrs.2016.02.020.

Ghanem, Carolina I., Swetha Rudraiah, Amy M. Bataille, María B. Vigo, Michael J. Goedken, and José E. Manautou. "Role of Nuclear Factor-Erythroid 2-Related Factor 2 (Nrf2) in the Transcriptional Regulation of Brain ABC Transporters during Acute Acetaminophen (APAP) Intoxication in Mice." *Biochemical Pharmacology* 94, no. 3 (April 1, 2015): 203–11. https://doi.org/10.1016/j.bcp.2015.01.013.

Giuffrida, Andrea, Massimiliano Beltramo, and Daniele Piomelli. "Mechanisms of Endocannabinoid Inactivation: Biochemistry and Pharmacology." *Journal of Pharmacology and Experimental Therapeutics* 1, no. 298 (2001).

Glasson, Emma J., Carol Bower, Beverly Petterson, Nick de Klerk, Gervase Chaney, and Joachim F. Hallmayer. "Perinatal Factors and the Development of Autism: A Population Study." *Archives of General Psychiatry* 61, no. 6 (June 1, 2004): 618–27. https://doi.org/10.1001/archpsyc.61.6.618.

Goines, Paula E., and Paul Ashwood. "Cytokine Dysregulation in Autism Spectrum Disorders (ASD): Possible Role of the Environment." *Neurotoxicology and Teratology* 36 (March 2013): 67–81. https://doi.org/10.1016/j.ntt.2012.07.006.

Golding, Jean, Steven Gregory, Rosie Clark, Genette Ellis, Yasmin Iles-Caven, and Kate Northstone. "Associations between Paracetamol (Acetaminophen) Intake between 18 and 32 Weeks Gestation and Neurocognitive Outcomes in the Child: A Longitudinal Cohort Study." *Paediatric and Perinatal Epidemiology* 34, no. 3 (May 2020): 257–66. https://doi.org/10.1111/ppe.12582.

Goldman, S. E., M. L. Alder, H. J. Burgess, B. A. Corbett, R. Hundley, D. Wofford, D. B. Fawkes, L. Wang, M. L. Laudenslager, and B. A. Malow. "Characterizing Sleep in

Adolescents and Adults with Autism Spectrum Disorders." *Journal of Autism and Developmental Disorders* 47, no. 6 (June 2017): 1682–95. https://doi.org/10.1007/s10803-017-3089-1.

Goodman, R., T. Ford, H. Richards, R. Gatward, and H. Meltzer. "The Development and Well-Being Assessment: Description and Initial Validation of an Integrated Assessment of Child and Adolescent Psychopathology." *Journal of Child Psychology and Psychiatry, and Allied Disciplines* 41, no. 5 (July 2000): 645–55.

Gotham, Katherine, Steven M. Brunwasser, and Catherine Lord. "Depressive and Anxiety Symptom Trajectories From School Age Through Young Adulthood in Samples With Autism Spectrum Disorder and Developmental Delay." *Journal of the American Academy of Child & Adolescent Psychiatry* 54, no. 5 (May 2015): 369-376.e3. https://doi.org/10.1016/j.jaac.2015.02.005.

Gotham, Katherine, Susan Risi, Geraldine Dawson, Helen Tager-Flusberg, Robert Joseph, Alice Carter, Susan Hepburn, et al. "A Replication of the Autism Diagnostic Observation Schedule (ADOS) Revised Algorithms." *Journal of the American Academy of Child & Adolescent Psychiatry* 47, no. 6 (June 2008): 642–51. https://doi.org/10.1097/CHI.0b013e31816bffb7.

Gou, Xiaoyun, Yan Wang, Ying Tang, Yi Qu, Jun Tang, Jing Shi, Dongqiong Xiao, and Dezhi Mu. "Association of Maternal Prenatal Acetaminophen Use with the Risk of Attention Deficit/Hyperactivity Disorder in Offspring: A Meta-Analysis." *Australian & New Zealand Journal of Psychiatry* 53, no. 3 (March 2019): 195–206. https://doi.org/10.1177/0004867418823276.

Gould, Georgianna G., Alexandre Seillier, Gabriela Weiss, Andrea Giuffrida, Teresa F. Burke, Julie G. Hensler, Crystal Rock, et al. "Acetaminophen Differentially Enhances Social Behavior and Cortical Cannabinoid Levels in Inbred Mice." *Progress in Neuro-Psychopharmacology and Biological Psychiatry* 38, no. 2 (August 2012): 260–69. https://doi.org/10.1016/j.pnpbp.2012.04.011.

Green, Joshua J., and Eric Hollander. "Autism and Oxytocin: New Developments in Translational Approaches to Therapeutics." *Neurotherapeutics* 7, no. 3 (July 2010): 250–57. https://doi.org/10.1016/j.nurt.2010.05.006.

Greenberg, B. D., U. Ziemann, G. Corá-Locatelli, A. Harmon, D. L. Murphy, J. C. Keel, and E. M. Wassermann. "Altered Cortical Excitability in Obsessive-Compulsive Disorder." *Neurology* 54, no. 1 (January 11, 2000): 142–47. https://doi.org/10.1212/wnl.54.1.142.

Greene, Ross W., Joseph Biederman, Stephen V. Faraone, Cheryl A. Ouellette, Courtney Penn, and Susan M. Griffin. "Toward a New Psychometric Definition of Social Disability in Children with Attention-Deficit Hyperactivity Disorder." *Journal of the American Academy of Child & Adolescent Psychiatry* 35, no. 5 (May 1996): 571–78. https://doi.org/10.1097/00004583-199605000-00011.

Greenlee, Jessica L., Angela S. Mosley, Amy M. Shui, Jeremy Veenstra-VanderWeele, and Katherine O. Gotham. "Medical and Behavioral Correlates of Depression History in Children and Adolescents With Autism Spectrum Disorder." *Pediatrics* 137, no.

Supplement_2 (February 1, 2016): S105–14. https://doi.org/10.1542/peds.2015-2851I.

Grether, Judith K., Meredith C. Anderson, Lisa A. Croen, Daniel Smith, and Gayle C. Windham. "Risk of Autism and Increasing Maternal and Paternal Age in a Large North American Population." *American Journal of Epidemiology* 170, no. 9 (November 1, 2009): 1118–26. https://doi.org/10.1093/aje/kwp247.

———. "Risk of Autism and Increasing Maternal and Paternal Age in a Large North American Population." *American Journal of Epidemiology* 170, no. 9 (November 1, 2009): 1118–26. https://doi.org/10.1093/aje/kwp247.

Grodberg, David, Paige M. Weinger, Danielle Halpern, Michael Parides, Alexander Kolevzon, and Joseph D. Buxbaum. "The Autism Mental Status Exam: Sensitivity and Specificity Using DSM-5 Criteria for Autism Spectrum Disorder in Verbally Fluent Adults." *Journal of Autism and Developmental Disorders* 44, no. 3 (March 2014): 609–14. https://doi.org/10.1007/s10803-013-1917-5.

Grzadzinski, Rebecca, Adriana Di Martino, Emily Brady, Maria Angeles Mairena, Matthew O'Neale, Eva Petkova, Catherine Lord, and F. Xavier Castellanos. "Examining Autistic Traits in Children with ADHD: Does the Autism Spectrum Extend to ADHD?" *Journal of Autism and Developmental Disorders* 41, no. 9 (September 2011): 1178–91. https://doi.org/10.1007/s10803-010-1135-3.

Grzadzinski, Rebecca, Catherine Dick, Catherine Lord, and Somer Bishop. "Parent-Reported and Clinician-Observed Autism Spectrum Disorder (ASD) Symptoms in Children with Attention Deficit/Hyperactivity Disorder (ADHD): Implications for Practice under DSM-5." *Molecular Autism* 7 (January 19, 2016): 7. https://doi.org/10.1186/s13229-016-0072-1.

———. "Parent-Reported and Clinician-Observed Autism Spectrum Disorder (ASD) Symptoms in Children with Attention Deficit/Hyperactivity Disorder (ADHD): Implications for Practice under DSM-5." *Molecular Autism* 7, no. 1 (December 2016): 7. https://doi.org/10.1186/s13229-016-0072-1.

Guastella, Adam J., Stewart L. Einfeld, Kylie M. Gray, Nicole J. Rinehart, Bruce J. Tonge, Timothy J. Lambert, and Ian B. Hickie. "Intranasal Oxytocin Improves Emotion Recognition for Youth with Autism Spectrum Disorders." *Biological Psychiatry* 67, no. 7 (April 2010): 692–94. https://doi.org/10.1016/j.biopsych.2009.09.020.

Gude, Neil M., Claire T. Roberts, Bill Kalionis, and Roger G. King. "Growth and Function of the Normal Human Placenta." *Thrombosis Research*, Special issue - State-of-the-Art 11th International Congress on Antiphospholipid Antibodies, 114, no. 5 (January 1, 2004): 397–407. https://doi.org/10.1016/j.thromres.2004.06.038.

Guilmatre, Audrey, Guillaume Huguet, Richard Delorme, and Thomas Bourgeron. "The Emerging Role of *SHANK* Genes in Neuropsychiatric Disorders: SHANK Genes in Neuropsychiatric Disorders." *Developmental Neurobiology* 74, no. 2 (February 2014): 113–22. https://doi.org/10.1002/dneu.22128.

Guo, Yu-Xiong, Hong-Xia Ma, Yu-Xin Zhang, Zhi-Hong Chen, and Qiong-Xiang Zhai. "Whole-Exome Sequencing for Identifying Genetic Causes of Intellectual Developmental Disorders." *International Journal of General Medicine* Volume 14 (April 2021): 1275–

82. https://doi.org/10.2147/IJGM.S300775.

Gupta, Surbhi, Andrea Caskey, Neelkamal Soares, and Marilyn Augustyn. "Autism Spectrum Disorder and Mental Health Comorbidity Leading to Prolonged Inpatient Admission." *Journal of Developmental & Behavioral Pediatrics* 39, no. 6 (July 2018): 523–25. https://doi.org/10.1097/DBP.0000000000000599.

Gustavson, Kristin, Helga Ask, Eivind Ystrom, Camilla Stoltenberg, W. Ian Lipkin, Pål Surén, Siri E. Håberg, et al. "Maternal Fever during Pregnancy and Offspring Attention Deficit Hyperactivity Disorder." *Scientific Reports* 9, no. 1 (December 2019): 9519. https://doi.org/10.1038/s41598-019-45920-7.

Gustavson, Kristin, Eivind Ystrom, Helga Ask, Fartein Ask Torvik, Mady Hornig, Ezra Susser, W. Ian Lipkin, et al. "Acetaminophen Use during Pregnancy and Offspring Attention Deficit Hyperactivity Disorder – a Longitudinal Sibling Control Study." *JCPP Advances* 1, no. 2 (July 2021). https://doi.org/10.1002/jcv2.12020.

Guthrie, Whitney, Lauren B. Swineford, Charly Nottke, and Amy M. Wetherby. "Early Diagnosis of Autism Spectrum Disorder: Stability and Change in Clinical Diagnosis and Symptom Presentation: Stability of Early Diagnosis and Symptoms in ASD." *Journal of Child Psychology and Psychiatry* 54, no. 5 (May 2013): 582–90. https://doi.org/10.1111/jcpp.12008.

Hack, Maureen, H. Gerry Taylor, Dennis Drotar, Mark Schluchter, Lydia Cartar, Laura Andreias, Deanne Wilson-Costello, and Nancy Klein. "Chronic Conditions, Functional Limitations, and Special Health Care Needs of School-Aged Children Born with Extremely Low-Birth-Weight in the 1990s." *JAMA* 294, no. 3 (July 20, 2005): 318–25. https://doi.org/10.1001/jama.294.3.318.

Hagmeyer, Simone, Jasmin Carmen Haderspeck, and Andreas Martin Grabrucker. "Behavioral Impairments in Animal Models for Zinc Deficiency." *Frontiers in Behavioral Neuroscience* 8 (January 6, 2015): 443. https://doi.org/10.3389/fnbeh.2014.00443.

Hall, Charlotte L., Boliang Guo, Althea Z. Valentine, Madeline J. Groom, David Daley, Kapil Sayal, and Chris Hollis. "The Validity of the Strengths and Difficulties Questionnaire (SDQ) for Children with ADHD Symptoms." *PLoS ONE* 14, no. 6 (June 19, 2019): e0218518. https://doi.org/10.1371/journal.pone.0218518.

Hallett, Victoria, Luc Lecavalier, Denis G. Sukhodolsky, Noreen Cipriano, Michael G. Aman, James T. McCracken, Christopher J. McDougle, et al. "Exploring the Manifestations of Anxiety in Children with Autism Spectrum Disorders." *Journal of Autism and Developmental Disorders* 43, no. 10 (October 2013): 2341–52. https://doi.org/10.1007/s10803-013-1775-1.

Hallmayer, Joachim, Sue Cleveland, Andrea Torres, Jennifer Phillips, Brianne Cohen, Tiffany Torigoe, Janet Miller, et al. "Genetic Heritability and Shared Environmental Factors Among Twin Pairs With Autism." *Archives of General Psychiatry* 68, no. 11 (November 2011): 1095–1102. https://doi.org/10.1001/archgenpsychiatry.2011.76.

Hammer, Rubi, Gillian E. Cooke, Mark A. Stein, and James R. Booth. "Functional Neuroimaging of Visuospatial Working Memory Tasks Enables Accurate Detection of Attention Deficit and Hyperactivity Disorder." *NeuroImage : Clinical* 9 (September 1, 2015): 244–52. https://doi.org/10.1016/j.nicl.2015.08.015.

Hansen, Richard, Susan H. Berry, Indrani Mukhopadhya, John M. Thomson, Karin A. Saunders, Charlotte E. Nicholl, W. Michael Bisset, et al. "The Microaerophilic Microbiota of De-Novo Paediatric Inflammatory Bowel Disease: The BISCUIT Study." Edited by Stefan Bereswill. *PLoS ONE* 8, no. 3 (March 12, 2013): e58825. https://doi.org/10.1371/journal.pone.0058825.

Happé, Francesca, Rhonda Booth, Rebecca Charlton, and Claire Hughes. "Executive Function Deficits in Autism Spectrum Disorders and Attention-Deficit/Hyperactivity Disorder: Examining Profiles across Domains and Ages." *Brain and Cognition*, Special Issue: The Cognitive Neuroscience of Asperger Syndrome, 61, no. 1 (June 1, 2006): 25–39. https://doi.org/10.1016/j.bandc.2006.03.004.

Hardan, Antonio Y., Lawrence K. Fung, Robin A. Libove, Tetyana V. Obukhanych, Surekha Nair, Leonore A. Herzenberg, Thomas W. Frazier, and Rabindra Tirouvanziam. "A Randomized Controlled Pilot Trial of Oral N-Acetylcysteine in Children with Autism." *Biological Psychiatry* 71, no. 11 (June 2012): 956–61. https://doi.org/10.1016/j.biopsych.2012.01.014.

Hart, Heledd, Joaquim Radua, Tomohiro Nakao, David Mataix-Cols, and Katya Rubia. "Meta-Analysis of Functional Magnetic Resonance Imaging Studies of Inhibition and Attention in Attention-Deficit/Hyperactivity Disorder: Exploring Task-Specific, Stimulant Medication, and Age Effects." *JAMA Psychiatry* 70, no. 2 (February 2013): 185–98. https://doi.org/10.1001/jamapsychiatry.2013.277.

Harvey, Louise, and Patricia Boksa. "Additive Effects of Maternal Iron Deficiency and Prenatal Immune Activation on Adult Behaviors in Rat Offspring." *Brain, Behavior, and Immunity* 40 (August 2014): 27–37. https://doi.org/10.1016/j.bbi.2014.06.005.

Hategan, Ana, James A. Bourgeois, and Jeremy Goldberg. "Aging with Autism Spectrum Disorder: An Emerging Public Health Problem." *International Psychogeriatrics* 29, no. 4 (April 2017): 695–97. https://doi.org/10.1017/S1041610216001599.

Hayashi, Wakaho, Yoichi Hanawa, Yuriko Iwami, Keisuke Aoyagi, Nobuyuki Saga, Dan Nakamura, and Akira Iwanami. "Correction to: ASD Symptoms in Adults with ADHD: A Preliminary Study Using the ADOS-2." *European Archives of Psychiatry and Clinical Neuroscience* 272, no. 2 (March 1, 2022): 233–233. https://doi.org/10.1007/s00406-021-01317-0.

Hayashi, Wakaho, Yoichi Hanawa, Nobuyuki Saga, Dan Nakamura, and Akira Iwanami. "ASD Symptoms in Adults with ADHD: A Comparative Study Using ADOS-2." *European Archives of Psychiatry and Clinical Neuroscience* 272, no. 8 (December 2022): 1481–94. https://doi.org/10.1007/s00406-021-01362-9.

Hazlett, Erin A., Monte S. Buchsbaum, Pauline Hsieh, M. Mehmet Haznedar, Jimcy Platholi, Elizabeth M. LiCalzi, Charles Cartwright, and Eric Hollander. "Regional Glucose Metabolism within Cortical Brodmann Areas in Healthy Individuals and Autistic Patients." *Neuropsychobiology* 49, no. 3 (2004): 115–25. https://doi.org/10.1159/000076719.

Haznedar, M Mehmet. "Volumetric Analysis and Three-Dimensional Glucose Metabolic Mapping of the Striatum and Thalamus in Patients With Autism Spectrum Disorders." *Am J Psychiatry*, 2006.

Haznedar, M. Mehmet, Monte S. Buchsbaum, Tse-Chung Wei, Patrick R. Hof, Charles Cartwright, Carol A. Bienstock, and Eric Hollander. "Limbic Circuitry in Patients With Autism Spectrum Disorders Studied With Positron Emission Tomography and Magnetic Resonance Imaging." *American Journal of Psychiatry* 157, no. 12 (December 2000): 1994–2001. https://doi.org/10.1176/appi.ajp.157.12.1994.

He, Huizhong, Ningxiao Ye, Lixin Yi, and Changjiang Yang. "Validating the Repetitive Behavior Scale-Revised for Children in China Aged 3 to 8 with Autism Spectrum Disorder." *Journal of Autism and Developmental Disorders* 49, no. 12 (December 2019): 4941–56. https://doi.org/10.1007/s10803-019-04210-x.

Hegvik, Tor-Arne, Qi Chen, Ralf Kuja-Halkola, Kari Klungsøyr, Agnieszka Butwicka, Paul Lichtenstein, Catarina Almqvist, Stephen V Faraone, Jan Haavik, and Henrik Larsson. "Familial Co-Aggregation of Attention-Deficit/Hyperactivity Disorder and Autoimmune Diseases: A Cohort Study Based on Swedish Population-Wide Registers." *International Journal of Epidemiology* 51, no. 3 (August 11, 2021): 898–909. https://doi.org/10.1093/ije/dyab151.

Hegvik, Tor-Arne, Johanne Telnes Instanes, Jan Haavik, Kari Klungsøyr, and Anders Engeland. "Associations between Attention-Deficit/Hyperactivity Disorder and Autoimmune Diseases Are Modified by Sex: A Population-Based Cross-Sectional Study." *European Child & Adolescent Psychiatry* 27, no. 5 (2018): 663–75. https://doi.org/10.1007/s00787-017-1056-1.

Heijnen-Kohl, S. M. J., R. M. Kok, R. M. H. J. Wilting, G. Rossi, and S. P. J. Van Alphen. "Screening of Autism Spectrum Disorders in Geriatric Psychiatry." *Journal of Autism and Developmental Disorders* 47, no. 9 (September 2017): 2679–89. https://doi.org/10.1007/s10803-017-3185-2.

Hill, Aj, Ms Mercier, Tdm Hill, Se Glyn, Na Jones, Y Yamasaki, T Futamura, et al. "Cannabidivarin Is Anticonvulsant in Mouse and Rat: Cannabidivarin as an Anticonvulsant." *British Journal of Pharmacology* 167, no. 8 (December 2012): 1629–42. https://doi.org/10.1111/j.1476-5381.2012.02207.x.

Hill, Andrew J., Claire M. Williams, Benjamin J. Whalley, and Gary J. Stephens. "Phytocannabinoids as Novel Therapeutic Agents in CNS Disorders." *Pharmacology & Therapeutics* 133, no. 1 (January 2012): 79–97. https://doi.org/10.1016/j.pharmthera.2011.09.002.

Hill, Austin Bradford. "The Environment and Disease: Association or Causation?" *Proceedings of the Royal Society of Medicine* 58, no. 5 (May 1965): 295–300.

Hill, T D M, M-G Cascio, B Romano, M Duncan, R G Pertwee, C M Williams, B J Whalley, and A J Hill. "Cannabidivarin-Rich Cannabis Extracts Are Anticonvulsant in Mouse and Rat via a CB $_1$ Receptor-Independent Mechanism: CBDV-Rich Extracts Are Anticonvulsant." *British Journal of Pharmacology* 170, no. 3 (October 2013): 679–92. https://doi.org/10.1111/bph.12321.

Hirsch, Lauren E, and Tamara Pringsheim. "Aripiprazole for Autism Spectrum Disorders (ASD)." Edited by Cochrane Developmental, Psychosocial and Learning Problems Group. *Cochrane Database of Systematic Reviews*, June 26, 2016. https://doi.org/10.1002/14651858.CD009043.pub3.

Hodgson, Anna R., Victoria Grahame, Deborah Garland, Fiona Gaultier, Jan Lecouturier, and Ann Le Couteur. "Parents' Opinions about an Intervention to Manage Repetitive Behaviours in Young Children with Autism Spectrum Disorder: A Qualitative Study." *Journal of Applied Research in Intellectual Disabilities* 31 (March 2018): 165–78. https://doi.org/10.1111/jar.12317.

Hofvander, Björn, Richard Delorme, Pauline Chaste, Agneta Nydén, Elisabet Wentz, Ola Ståhlberg, Evelyn Herbrecht, et al. "Psychiatric and Psychosocial Problems in Adults with Normal-Intelligence Autism Spectrum Disorders." *BMC Psychiatry* 9, no. 1 (December 2009): 35. https://doi.org/10.1186/1471-244X-9-35.

Hollander, E. "The Relationship between Repetitive Behaviors and Growth Hormone Response to Sumatriptan Challenge in Adult Autistic Disorder." *Neuropsychopharmacology* 22, no. 2 (February 2000): 163–67. https://doi.org/10.1016/S0893-133X(99)00121-9.

Hollander, Eric, Evdokia Anagnostou, William Chaplin, Katherine Esposito, M. Mehmet Haznedar, Elizabeth Licalzi, Stacey Wasserman, Latha Soorya, and Monte Buchsbaum. "Striatal Volume on Magnetic Resonance Imaging and Repetitive Behaviors in Autism." *Biological Psychiatry* 58, no. 3 (August 2005): 226–32. https://doi.org/10.1016/j.biopsych.2005.03.040.

Hollander, Eric, Jennifer Bartz, William Chaplin, Ann Phillips, Jennifer Sumner, Latha Soorya, Evdokia Anagnostou, and Stacey Wasserman. "Oxytocin Increases Retention of Social Cognition in Autism." *Biological Psychiatry* 61, no. 4 (February 2007): 498–503. https://doi.org/10.1016/j.biopsych.2006.05.030.

Hollander, Eric, William Chaplin, Latha Soorya, Stacey Wasserman, Sherry Novotny, Jade Rusoff, Nicole Feirsen, Lauren Pepa, and Evdokia Anagnostou. "Divalproex Sodium vs Placebo for the Treatment of Irritability in Children and Adolescents with Autism Spectrum Disorders." *Neuropsychopharmacology* 35, no. 4 (March 2010): 990–98. https://doi.org/10.1038/npp.2009.202.

Hollander, Eric, Gina DelGiudice-Asch, Lorraine Simon, James Schmeidler, Charles Cartwright, Concetta M. DeCaria, Jee Kwon, Charlotte Cunningham-Rundles, Floresta Chapman, and John B. Zabriskie. "B Lymphocyte Antigen D8/17 and Repetitive Behaviors in Autism." *American Journal of Psychiatry* 156, no. 2 (February 1, 1999): 317–20.

https://doi.org/10.1176/ajp.156.2.317.

Hollander, Eric, Rima Dolgoff-Kaspar, Charles Cartwright, Ronald Rawitt, and Sherie Novotny. "An Open Trial of Divalproex Sodium in Autism Spectrum Disorders." *The Journal of Clinical Psychiatry* 62, no. 7 (July 15, 2001): 530–34. https://doi.org/10.4088/JCP.v62n07a05.

Hollander, Eric, Suma Jacob, Roger Jou, Nora McNamara, Linmarie Sikich, Russell Tobe, Janice Smith, et al. "Balovaptan vs Placebo for Social Communication in Childhood Autism Spectrum Disorder: A Randomized Clinical Trial." *JAMA Psychiatry* 79, no. 8 (August 1, 2022): 760. https://doi.org/10.1001/jamapsychiatry.2022.1717.

Hollander, Eric, Alicia Kaplan, Charles Cartwright, and Daniel Reichman. "Venlafaxine in Children, Adolescents, and Young Adults With Autism Spectrum Disorders: An Open Retrospective Clinical Report." *Journal of Child Neurology* 15, no. 2 (February 2000): 132–35. https://doi.org/10.1177/088307380001500214.

Hollander, Eric, Suah Kim, Ashley Braun, Daphne Simeon, and Joseph Zohar. "Cross-Cutting Issues and Future Directions for the OCD Spectrum." *Psychiatry Research* 170, no. 1 (November 2009): 3–6. https://doi.org/10.1016/j.psychres.2008.07.015.

Hollander, Eric, Audrey King, Katherine Delaney, Christopher J Smith, and Jeremy M Silverman. "Obsessive–Compulsive Behaviors in Parents of Multiplex Autism Families." *Psychiatry Research* 117, no. 1 (January 2003): 11–16. https://doi.org/10.1016/S0165-1781(02)00304-9.

Hollander, Eric, Sherie Novotny, Margaret Hanratty, Rona Yaffe, Concetta M DeCaria, Bonnie R Aronowitz, and Serge Mosovich. "Oxytocin Infusion Reduces Repetitive Behaviors in Adults with Autistic and Asperger's Disorders." *Neuropsychopharmacology* 28, no. 1 (January 2003): 193–98. https://doi.org/10.1038/sj.npp.1300021.

Hollander, Eric, Ann Phillips, William Chaplin, Karen Zagursky, Sherie Novotny, Stacey Wasserman, and Rupa Iyengar. "A Placebo Controlled Crossover Trial of Liquid Fluoxetine on Repetitive Behaviors in Childhood and Adolescent Autism." *Neuropsychopharmacology* 30, no. 3 (March 2005): 582–89. https://doi.org/10.1038/sj.npp.1300627.

Hollander, Eric, and Joseph Klingenstein Professor. "New Developments in Autism Spectrum Disorders," n.d.

Hollander, Eric, Latha Soorya, William Chaplin, Evdokia Anagnostou, Bonnie P Taylor, Casara J Ferretti, Stacey Wasserman, Erika Swanson, and Cara Settipani. "A D o Ub Le-B Lind P Laceb o -Co Ntro Lled Trial o f Fluoxetine for Repetitive Behaviors and G Lobal Severity in A Dult A Utism Spectrum D Isorders." *Am J Psychiatry*, n.d.

Hollander, Eric, Latha Soorya, Stacey Wasserman, Katherine Esposito, William Chaplin, and Evdokia Anagnostou. "Divalproex Sodium vs. Placebo in the Treatment of Repetitive Behaviours in Autism Spectrum Disorder." *The International Journal of Neuropsychopharmacology* 9, no. 02 (August 15, 2005): 209. https://doi.org/10.1017/S1461145705005791.

Hollander, Eric, Genoveva Uzunova, Bonnie P. Taylor, Rachel Noone, Emma Racine, Ellen Doernberg, Katherine Freeman, and Casara Jean Ferretti. "Randomized Crossover Feasibility Trial of Helminthic *Trichuris Suis* Ova versus Placebo for Repetitive Behaviors in Adult Autism Spectrum Disorder." *The World Journal of Biological Psychiatry* 21, no. 4 (April 20, 2020): 291–99. https://doi.org/10.1080/15622975.2018.1523561.

Hoogman, Martine, Janita Bralten, Derrek P. Hibar, Maarten Mennes, Marcel P. Zwiers, Lizanne Schweren, Kimm J.E. van Hulzen, et al. "Subcortical Brain Volume Differences of Participants with ADHD across the Lifespan: An ENIGMA Collaboration." *The Lancet. Psychiatry* 4, no. 4 (April 2017): 310–19. https://doi.org/10.1016/S2215-0366(17)30049-4.

Hoogman, Martine, Ryan Muetzel, Joao P. Guimaraes, Elena Shumskaya, Maarten Mennes, Marcel P. Zwiers, Neda Jahanshad, et al. "Brain Imaging of the Cortex in ADHD: A Coordinated Analysis of Large-Scale Clinical and Population-Based Samples." *The American Journal of Psychiatry* 176, no. 7 (July 1, 2019): 531–42. https://doi.org/10.1176/appi.ajp.2019.18091033.

Hooker, Jessica L., Deanna Dow, Lindee Morgan, Christopher Schatschneider, and Amy M. Wetherby. "Psychometric Analysis of the Repetitive Behavior Scale-revised Using Confirmatory Factor Analysis in Children with Autism." *Autism Research* 12, no. 9 (September 2019): 1399–1410. https://doi.org/10.1002/aur.2159.

Hossain, Md Mahbub, Nusrat Khan, Abida Sultana, Ping Ma, E. Lisako J. McKyer, Helal Uddin Ahmed, and Neetu Purohit. "Prevalence of Comorbid Psychiatric Disorders among People with Autism Spectrum Disorder: An Umbrella Review of Systematic Reviews and Meta-Analyses." *Psychiatry Research* 287 (May 2020): 112922. https://doi.org/10.1016/j.psychres.2020.112922.

Howe, Stephanie J., Katie Hewitt, Jessica Baraskewich, Sarah Cassidy, and Carly A. McMorris. "Suicidality Among Children and Youth With and Without Autism Spectrum Disorder: A Systematic Review of Existing Risk Assessment Tools." *Journal of Autism and Developmental Disorders* 50, no. 10 (October 2020): 3462–76. https://doi.org/10.1007/s10803-020-04394-7.

Hsiao, Elaine Y., Sara W. McBride, Janet Chow, Sarkis K. Mazmanian, and Paul H. Patterson. "Modeling an Autism Risk Factor in Mice Leads to Permanent Immune Dysregulation." *Proceedings of the National Academy of Sciences* 109, no. 31 (July 31, 2012): 12776–81. https://doi.org/10.1073/pnas.1202556109.

Hsiao, Elaine Y., Sara W. McBride, Sophia Hsien, Gil Sharon, Embriette R. Hyde, Tyler McCue, Julian A. Codelli, et al. "The Microbiota Modulates Gut Physiology and Behavioral Abnormalities Associated with Autism." *Cell* 155, no. 7 (December 19, 2013): 1451–63. https://doi.org/10.1016/j.cell.2013.11.024.

Hsiao, Elaine Y., and Paul H. Patterson. "Activation of the Maternal Immune System Induces Endocrine Changes in the Placenta via IL-6." *Brain, Behavior, and Immunity* 25, no. 4 (May 2011): 604–15. https://doi.org/10.1016/j.bbi.2010.12.017.

Huang, Eric. "Neurotrophins: Roles in Neuronal Development and Function - PMC." Accessed March 23, 2023. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2758233/.

Huang, Jing-Syuan, Fu-Chi Yang, Wu-Chien Chien, Ta-Chuan Yeh, Chi-Hsiang Chung, Chia-Kuang Tsai, Shih-Jen Tsai, et al. "Risk of Substance Use Disorder and Its Associations With Comorbidities and Psychotropic Agents in Patients With Autism." *JAMA Pediatrics* 175, no. 2 (February 1, 2021): e205371. https://doi.org/10.1001/jamapediatrics.2020.5371.

Hultman, Christina M., Pär Sparén, and Sven Cnattingius. "Perinatal Risk Factors for Infantile Autism." *Epidemiology (Cambridge, Mass.)* 13, no. 4 (July 2002): 417–23. https://doi.org/10.1097/00001648-200207000-00009.

Hus, Vanessa, and Catherine Lord. "Effects of Child Characteristics on the Autism Diagnostic Interview-Revised: Implications for Use of Scores as a Measure of ASD Severity." *Journal of Autism and Developmental Disorders* 43, no. 2 (February 2013): 371–81. https://doi.org/10.1007/s10803-012-1576-y.

Hus, Vanessa, Andrew Pickles, Edwin H. Cook, Susan Risi, and Catherine Lord. "Using the Autism Diagnostic Interview—Revised to Increase Phenotypic Homogeneity in Genetic Studies of Autism." *Biological Psychiatry* 61, no. 4 (February 2007): 438–48. https://doi.org/10.1016/j.biopsych.2006.08.044.

Hwang, In-seong, and Soon-Beom Hong. "Association between Body Mass Index and Subcortical Volume in Pre-Adolescent Children with Autism Spectrum Disorder: An Exploratory Study." *Autism Research* 15, no. 12 (2022): 2238–49. https://doi.org/10.1002/aur.2834.

Iannotti, Fabio Arturo, Charlotte L. Hill, Antonio Leo, Ahlam Alhusaini, Camille Soubrane, Enrico Mazzarella, Emilio Russo, Benjamin J. Whalley, Vincenzo Di Marzo, and Gary J. Stephens. "Nonpsychotropic Plant Cannabinoids, Cannabidivarin (CBDV) and Cannabidiol (CBD), Activate and Desensitize Transient Receptor Potential Vanilloid 1 (TRPV1) Channels in Vitro: Potential for the Treatment of Neuronal Hyperexcitability." *ACS Chemical Neuroscience* 5, no. 11 (November 19, 2014): 1131–41. https://doi.org/10.1021/cn5000524.

International Classification of Diseases, Ninth Revision, March 3, 2022, https://www.cdc.gov/nchs/icd/icd9.htm.

International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM), October 17, 2022. https://www.cdc.gov/nchs/icd/icd-10-cm.htm.

International Classification of Diseases, Eleventh Revision for Mortality and Morbidity Statistics. Accessed June 13, 2023. https://icd.who.int/browse11/l-m/en.

Ikonomidou, Chrysanthy, and Angela M. Kaindl. "Neuronal Death and Oxidative Stress in the Developing Brain." *Antioxidants & Redox Signaling* 14, no. 8 (April 15, 2011): 1535–50. https://doi.org/10.1089/ars.2010.3581.

Inoue, Kosuke, Beate Ritz, Andreas Ernst, Wan-Ling Tseng, Yuying Yuan, Qi Meng, Cecilia Høst Ramlau-Hansen, et al. "Behavioral Problems at Age 11 Years After Prenatal and

Postnatal Exposure to Acetaminophen: Parent-Reported and Self-Reported Outcomes." *American Journal of Epidemiology* 190, no. 6 (June 1, 2021): 1009–20. https://doi.org/10.1093/aje/kwaa257.

Instanes, Johanne T., Anne Halmøy, Anders Engeland, Jan Haavik, Kari Furu, and Kari Klungsøyr. "Attention-Deficit/Hyperactivity Disorder in Offspring of Mothers With Inflammatory and Immune System Diseases." *Biological Psychiatry* 81, no. 5 (March 1, 2017): 452–59. https://doi.org/10.1016/j.biopsych.2015.11.024.

Ito, Hiroshi T., Stephen E.P. Smith, Elaine Hsiao, and Paul H. Patterson. "Maternal Immune Activation Alters Nonspatial Information Processing in the Hippocampus of the Adult Offspring." *Brain, Behavior, and Immunity* 24, no. 6 (August 2010): 930–41. https://doi.org/10.1016/j.bbi.2010.03.004.

Jacob, Suma, Evdokia Anagnostou, Eric Hollander, Roger Jou, Nora McNamara, Linmarie Sikich, Russell Tobe, et al. "Large Multicenter Randomized Trials in Autism: Key Insights Gained from the Balovaptan Clinical Development Program." *Molecular Autism* 13, no. 1 (December 2022): 25. https://doi.org/10.1186/s13229-022-00505-6.

Jaeschke, Hartmut, Olamide B. Adelusi, and Anup Ramachandran. "Ferroptosis and Acetaminophen Hepatotoxicity: Are We Going Down Another Rabbit Hole?" *Gene Expression* 20, no. 3 (June 11, 2021): 169–78. https://doi.org/10.3727/105221621X16104581979144.

Jaeschke, Hartmut, Jephte Y. Akakpo, David S. Umbaugh, and Anup Ramachandran. "Novel Therapeutic Approaches Against Acetaminophen-Induced Liver Injury and Acute Liver Failure." *Toxicological Sciences* 174, no. 2 (April 2020): 159. https://doi.org/10.1093/toxsci/kfaa002.

Jager-Hyman, Shari, Brenna B. Maddox, Samantha R. Crabbe, and David S. Mandell. "Mental Health Clinicians' Screening and Intervention Practices to Reduce Suicide Risk in Autistic Adolescents and Adults." *Journal of Autism and Developmental Disorders* 50, no. 10 (October 2020): 3450–61. https://doi.org/10.1007/s10803-020-04441-3.

Jakovcevski, Mira, and Schahram Akbarian. "Epigenetic Mechanisms in Neurodevelopmental and Neurodegenerative Disease." *Nature Medicine* 18, no. 8 (August 2012): 1194–1204. https://doi.org/10.1038/nm.2828.

James, S. Jill, Paul Cutler, Stepan Melnyk, Stefanie Jernigan, Laurette Janak, David W. Gaylor, and James A. Neubrander. "Metabolic Biomarkers of Increased Oxidative Stress and Impaired Methylation Capacity in Children with Autism." *The American Journal of Clinical Nutrition* 80, no. 6 (December 2004): 1611–17. https://doi.org/10.1093/ajcn/80.6.1611.

James, S. Jill, Stepan Melnyk, Stefanie Jernigan, Mario A. Cleves, Charles H. Halsted, Donna H. Wong, Paul Cutler, et al. "Metabolic Endophenotype and Related Genotypes Are Associated with Oxidative Stress in Children with Autism." *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics : The Official Publication of the International Society of Psychiatric Genetics* 141B, no. 8 (December 5, 2006): 947–56. https://doi.org/10.1002/ajmg.b.30366.

Jamison, Rene, Somer L Bishop, Marisela Huerta, and Alycia K Halladay. "The Clinician Perspective on Sex Differences in Autism Spectrum Disorders." *Autism* 21, no. 6 (August 2017): 772–84. https://doi.org/10.1177/1362361316681481.

Janecka, Magdalena, Stefan N. Hansen, Amirhossein Modabbernia, Heidi A. Browne, Joseph D. Buxbaum, Diana E. Schendel, Abraham Reichenberg, Erik T. Parner, and Dorothy E. Grice. "Parental Age and Differential Estimates of Risk for Neuropsychiatric Disorders: Findings From the Danish Birth Cohort." *Journal of the American Academy of Child and Adolescent Psychiatry* 58, no. 6 (June 2019): 618–27. https://doi.org/10.1016/j.jaac.2018.09.447.

Janssen, Aron, Howard Huang, and Christina Duncan. "Gender Variance Among Youth with Autism Spectrum Disorders: A Retrospective Chart Review." *Transgender Health* 1, no. 1 (February 1, 2016): 63–68. https://doi.org/10.1089/trgh.2015.0007.

Jean-Gilles, Lucie, Bruno Gran, and Cris S. Constantinescu. "Interaction between Cytokines, Cannabinoids and the Nervous System." *Immunobiology*, Special Issue: Cannabinoids and Immunology, 215, no. 8 (August 1, 2010): 606–10. https://doi.org/10.1016/j.imbio.2009.12.006.

Jeong, Hyeonsoo, Isabel Mendizabal, Stefano Berto, Paramita Chatterjee, Thomas Layman, Noriyoshi Usui, Kazuya Toriumi, et al. "Evolution of DNA Methylation in the Human Brain." *Nature Communications* 12, no. 1 (April 1, 2021): 2021. https://doi.org/10.1038/s41467-021-21917-7.

Jetten, Marlon J. A., Ainhoa Ruiz-Aracama, Maarten L. J. Coonen, Sandra M. Claessen, Marcel H. M. van Herwijnen, Arjen Lommen, Joost H. M. van Delft, Ad A. C. M. Peijnenburg, and Jos C. S. Kleinjans. "Interindividual Variation in Gene Expression Responses and Metabolite Formation in Acetaminophen-Exposed Primary Human Hepatocytes." *Archives of Toxicology* 90 (2016): 1103–15. https://doi.org/10.1007/s00204-015-1545-2.

Jetten, Marlon J.A., Stan Gaj, Ainhoa Ruiz-Aracama, Theo M. de Kok, Joost H.M. van Delft, Arjen Lommen, Eugene P. van Someren, et al. "'Omics Analysis of Low Dose Acetaminophen Intake Demonstrates Novel Response Pathways in Humans." *Toxicology and Applied Pharmacology* 259, no. 3 (March 2012): 320–28. https://doi.org/10.1016/j.taap.2012.01.009.

Ji, Yuelong, Romuladus E. Azuine, Yan Zhang, Wenpin Hou, Xiumei Hong, Guoying Wang, Anne Riley, Colleen Pearson, Barry Zuckerman, and Xiaobin Wang. "Association of Cord Plasma Biomarkers of In Utero Acetaminophen Exposure With Risk of Attention-Deficit/Hyperactivity Disorder and Autism Spectrum Disorder in Childhood." *JAMA Psychiatry* 77, no. 2 (February 1, 2020): 180. https://doi.org/10.1001/jamapsychiatry.2019.3259.

Ji, Yuelong, Anne W Riley, Li-Ching Lee, Xiumei Hong, Guoying Wang, Hui-Ju Tsai, Noel T Mueller, et al. "Maternal Biomarkers of Acetaminophen Use and Offspring Attention Deficit Hyperactivity Disorder." *Brain Sciences* 8, no. 127 (2018): 15. https://doi.org/doi:10.3390/brainsci8070127.

Jia, Ronnie, Zachary R. Steelman, and Heather H. Jia. “Psychometric Assessments of Three Self-Report Autism Scales (AQ, RBQ-2A, and SQ) for General Adult Populations.” *Journal of Autism and Developmental Disorders* 49, no. 5 (May 2019): 1949–65. https://doi.org/10.1007/s10803-019-03880-x.

Jiménez, Elizabeth Carolina, Claudia Avella-Garcia, James Kustow, Sally Cubbin, Montse Corrales, Vanessa Richarte, Flavia Lorena Esposito, et al. “Eye Vergence Responses During an Attention Task in Adults With ADHD and Clinical Controls.” *Journal of Attention Disorders* 25, no. 9 (July 2021): 1302–10. https://doi.org/10.1177/1087054719897806.

Jiujias, Marina, Elizabeth Kelley, and Layla Hall. “Restricted, Repetitive Behaviors in Autism Spectrum Disorder and Obsessive–Compulsive Disorder: A Comparative Review.” *Child Psychiatry & Human Development* 48, no. 6 (December 2017): 944–59. https://doi.org/10.1007/s10578-017-0717-0.

Johnston, Krista Haley Smith, and Grace Iarocci. “Are Generalized Anxiety and Depression Symptoms Associated with Social Competence in Children with and without Autism Spectrum Disorder?” *Journal of Autism and Developmental Disorders* 47, no. 12 (December 2017): 3778–88. https://doi.org/10.1007/s10803-017-3056-x.

Jokiranta, Elina, Alan S. Brown, Markus Heinimaa, Keely Cheslack-Postava, Auli Suominen, and Andre Sourander. “Parental Psychiatric Disorders and Autism Spectrum Disorders.” *Psychiatry Research* 207, no. 3 (May 2013): 203–11. https://doi.org/10.1016/j.psychres.2013.01.005.

Jokiranta-Olkoniemi, Elina, Keely Cheslack-Postava, Dan Sucksdorff, Auli Suominen, David Gyllenberg, Roshan Chudal, Susanna Leivonen, Mika Gissler, Alan S. Brown, and Andre Sourander. “Risk of Psychiatric and Neurodevelopmental Disorders Among Siblings of Probands With Autism Spectrum Disorders.” *JAMA Psychiatry* 73, no. 6 (June 1, 2016): 622–29. https://doi.org/10.1001/jamapsychiatry.2016.0495.

Joseph, Nidhin, Yanli Zhang-James, Andras Perl, and Stephen V. Faraone. “Oxidative Stress and ADHD: A Meta-Analysis.” *Journal of Attention Disorders* 19, no. 11 (November 2015): 915–24. https://doi.org/10.1177/1087054713510354.

Josephson, Stephen C., Eric Hollander, and Jennifer Sumner. “Comprehensive Treatment of Three Patients with Comorbid OCPD and ADHD.” *CNS Spectrums* 12, no. 5 (May 2007): 338–42. https://doi.org/10.1017/S1092852900021131.

Joshi, Gagan, Carter Petty, Janet Wozniak, Aude Henin, Ronna Fried, Maribel Galdo, Meghan Kotarski, Sarah Walls, and Joseph Biederman. “The Heavy Burden of Psychiatric Comorbidity in Youth with Autism Spectrum Disorders: A Large Comparative Study of a Psychiatrically Referred Population.” *Journal of Autism and Developmental Disorders* 40, no. 11 (November 2010): 1361–70. https://doi.org/10.1007/s10803-010-0996-9.

Joyce, Caroline, Emma Honey, Susan R. Leekam, Sarah L. Barrett, and Jacqui Rodgers. “Anxiety, Intolerance of Uncertainty and Restricted and Repetitive Behaviour: Insights Directly from Young People with ASD.” *Journal of Autism and Developmental Disorders* 47, no. 12 (December 2017): 3789–3802. https://doi.org/10.1007/s10803-017-

3027-2.

Jung, Kwang-Mook, Marja Sepers, Christopher M. Henstridge, Olivier Lassalle, Daniela Neuhofer, Henry Martin, Melanie Ginger, et al. "Uncoupling of the Endocannabinoid Signalling Complex in a Mouse Model of Fragile X Syndrome." *Nature Communications* 3 (2012): 1080. https://doi.org/10.1038/ncomms2045.

Jyonouchi, Harumi, Lee Geng, Deanna L. Streck, and Gokce A. Toruner. "Children with Autism Spectrum Disorders (ASD) Who Exhibit Chronic Gastrointestinal (GI) Symptoms and Marked Fluctuation of Behavioral Symptoms Exhibit Distinct Innate Immune Abnormalities and Transcriptional Profiles of Peripheral Blood (PB) Monocytes." *Journal of Neuroimmunology* 238, no. 1–2 (September 2011): 73–80. https://doi.org/10.1016/j.jneuroim.2011.07.001.

Kaat, Aaron J., Amy M. Shui, Sheila S. Ghods, Cristan A. Farmer, Amy N. Esler, Audrey Thurm, Stelios Georgiades, et al. "Sex Differences in Scores on Standardized Measures of Autism Symptoms: A Multisite Integrative Data Analysis." *Journal of Child Psychology and Psychiatry* 62, no. 1 (January 2021): 97–106. https://doi.org/10.1111/jcpp.13242.

Kajta, Małgorzata, and Anna K. Wójtowicz. "Impact of Endocrine-Disrupting Chemicals on Neural Development and the Onset of Neurological Disorders." *Pharmacological Reports: PR* 65, no. 6 (2013): 1632–39. https://doi.org/10.1016/s1734-1140(13)71524-x.

Kaltenegger, Helena C., Björn Philips, and Peter Wennberg. "Autistic Traits in Mentalization-based Treatment for Concurrent Borderline Personality Disorder and Substance Use Disorder: Secondary Analyses of a Randomized Controlled Feasibility Study." *Scandinavian Journal of Psychology* 61, no. 3 (June 2020): 416–22. https://doi.org/10.1111/sjop.12595.

Kapellen, Thomas M., Rebecca Reimann, Wieland Kiess, and Karel Kostev. "Prevalence of Medically Treated Children with ADHD and Type 1 Diabetes in Germany - Analysis of Two Representative Databases." *Journal of Pediatric Endocrinology & Metabolism: JPEM* 29, no. 11 (November 1, 2016): 1293–97. https://doi.org/10.1515/jpem-2016-0171.

Karhson, Debra S., Karolina M. Krasinska, Jamie Ahloy Dallaire, Robin A. Libove, Jennifer M. Phillips, Allis S. Chien, Joseph P. Garner, Antonio Y. Hardan, and Karen J. Parker. "Plasma Anandamide Concentrations Are Lower in Children with Autism Spectrum Disorder." *Molecular Autism* 9, no. 1 (December 2018): 18. https://doi.org/10.1186/s13229-018-0203-y.

Keil, Alexander, Julie L. Daniels, Ulla Forssen, Christina Hultman, Sven Cnattingius, Karin C. Söderberg, Maria Feychting, and Par Sparen. "Parental Autoimmune Diseases Associated With Autism Spectrum Disorders in Offspring." *Epidemiology* 21, no. 6 (November 2010): 805–8. https://doi.org/10.1097/EDE.0b013e3181f26e3f.

Kenworthy, Lauren, Laura Gutermuth Anthony, Daniel Q. Naiman, Lynn Cannon, Meagan C. Wills, Caroline Luong-Tran, Monica Adler Werner, et al. "Randomized Controlled Effectiveness Trial of Executive Function Intervention for Children on the Autism

Spectrum." *Journal of Child Psychology and Psychiatry* 55, no. 4 (April 2014): 374–83. https://doi.org/10.1111/jcpp.12161.

Kerr, D. M., L. Downey, M. Conboy, D. P. Finn, and M. Roche. "Alterations in the Endocannabinoid System in the Rat Valproic Acid Model of Autism." *Behavioural Brain Research* 249 (July 15, 2013): 124–32. https://doi.org/10.1016/j.bbr.2013.04.043.

Kian, Naghmeh, Noosha Samieefar, and Nima Rezaei. "Prenatal Risk Factors and Genetic Causes of ADHD in Children." *World Journal of Pediatrics* 18, no. 5 (May 2022): 308–19. https://doi.org/10.1007/s12519-022-00524-6.

Kim, Jae Han, Jong Yeob Kim, Jinhee Lee, Gwang Hun Jeong, Eun Lee, San Lee, Keum Hwa Lee, et al. "Environmental Risk Factors, Protective Factors, and Peripheral Biomarkers for ADHD: An Umbrella Review." *The Lancet Psychiatry* 7, no. 11 (November 1, 2020): 955–70. https://doi.org/10.1016/S2215-0366(20)30312-6.

Kim, Jong Yeob, Min Ji Son, Chei Yun Son, Joaquim Radua, Michael Eisenhut, Florence Gressier, Ai Koyanagi, et al. "Environmental Risk Factors and Biomarkers for Autism Spectrum Disorder: An Umbrella Review of the Evidence." *The Lancet Psychiatry* 6, no. 7 (July 2019): 590–600. https://doi.org/10.1016/S2215-0366(19)30181-6.

Kim, So Hyun, Rebecca Grzadzinski, Kassandra Martinez, and Catherine Lord. "Measuring Treatment Response in Children with Autism Spectrum Disorder: Applications of the Brief Observation of Social Communication Change to the Autism Diagnostic Observation Schedule." *Autism* 23, no. 5 (July 2019): 1176–85. https://doi.org/10.1177/1362361318793253.

King, Bryan H. "Fluoxetine and Repetitive Behaviors in Children and Adolescents With Autism Spectrum Disorder." *JAMA* 322, no. 16 (October 22, 2019): 1557. https://doi.org/10.1001/jama.2019.11738.

King, Bryan H., Nina De Lacy, and Matthew Siegel. "Psychiatric Assessment of Severe Presentations in Autism Spectrum Disorders and Intellectual Disability." *Child and Adolescent Psychiatric Clinics of North America* 23, no. 1 (January 2014): 1–14. https://doi.org/10.1016/j.chc.2013.07.001.

King, Bryan H., Kimberly Dukes, Craig L. Donnelly, Linmarie Sikich, James T. McCracken, Lawrence Scahill, Eric Hollander, et al. "Baseline Factors Predicting Placebo Response to Treatment in Children and Adolescents With Autism Spectrum Disorders: A Multisite Randomized Clinical Trial." *JAMA Pediatrics* 167, no. 11 (November 1, 2013): 1045. https://doi.org/10.1001/jamapediatrics.2013.2698.

King, Bryan H., Eric Hollander, Linmarie Sikich, James T. McCracken, Lawrence Scahill, Joel D. Bregman, Craig L. Donnelly, et al. "Lack of Efficacy of Citalopram in Children With Autism Spectrum Disorders and High Levels of Repetitive Behavior: Citalopram Ineffective in Children With Autism." *Archives of General Psychiatry* 66, no. 6 (June 1, 2009): 583. https://doi.org/10.1001/archgenpsychiatry.2009.30.

King, Bryan H, D Mark Wright, Benjamin L Handen, Linmarie Sikich, Andrew W Zimmerman, William Mcmahon, Erin Cantwell, et al. "Double-Blind, Placebo-Controlled Study of Amantadine Hydrochloride in the Treatment of Children With Autistic Disorder." *J. AM. ACAD. CHILD ADOLESC. PSYCHIATRY*, 2001.

Kirsten, Thiago B., Luciana L. Lippi, Estela Bevilacqua, and Maria M. Bernardi. "LPS Exposure Increases Maternal Corticosterone Levels, Causes Placental Injury and Increases IL-1B Levels in Adult Rat Offspring: Relevance to Autism." Edited by Muriel Moser. *PLoS ONE* 8, no. 12 (December 2, 2013): e82244. https://doi.org/10.1371/journal.pone.0082244.

Klein, Rodrigo Moreno, Camila Rigobello, Camila Borecki Vidigal, Kawane Fabrício Moura, Décio Sabbatini Barbosa, Daniela Cristina Ceccatto Gerardin, Graziela Scalianti Ceravolo, and Estefânia Gastaldello Moreira. "Gestational Exposure to Paracetamol in Rats Induces Neurofunctional Alterations in the Progeny." *Neurotoxicology and Teratology* 77 (January 2020): 106838. https://doi.org/10.1016/j.ntt.2019.106838.

Klein, Thomas W., and Guy A. Cabral. "Cannabinoid-Induced Immune Suppression and Modulation of Antigen-Presenting Cells." *Journal of Neuroimmune Pharmacology* 1, no. 1 (March 1, 2006): 50–64. https://doi.org/10.1007/s11481-005-9007-x.

Klinger-Gratz, Pascal P., William T. Ralvenius, Elena Neumann, Ako Kato, Rita Nyilas, Zsolt Lele, István Katona, and Hanns Ulrich Zeilhofer. "Acetaminophen Relieves Inflammatory Pain through CB1 Cannabinoid Receptors in the Rostral Ventromedial Medulla." *The Journal of Neuroscience* 38, no. 2 (January 10, 2018): 322–34. https://doi.org/10.1523/JNEUROSCI.1945-17.2017.

Kobayashi, Tohru, Tasuku Matsuyama, Masanobu Takeuchi, and Shinya Ito. "Autism Spectrum Disorder and Prenatal Exposure to Selective Serotonin Reuptake Inhibitors: A Systematic Review and Meta-Analysis." *Reproductive Toxicology* 65 (October 2016): 170–78. https://doi.org/10.1016/j.reprotox.2016.07.016.

Koegel, Lynn Kern, Mi N. Park, and Robert L. Koegel. "Using Self-Management to Improve the Reciprocal Social Conversation of Children with Autism Spectrum Disorder." *Journal of Autism and Developmental Disorders* 44, no. 5 (May 2014): 1055–63. https://doi.org/10.1007/s10803-013-1956-y.

Kohane, Isaac S. "An Autism Case History to Review the Systematic Analysis of Large-Scale Data to Refine the Diagnosis and Treatment of Neuropsychiatric Disorders." *Biological Psychiatry* 77, no. 1 (January 2015): 59–65. https://doi.org/10.1016/j.biopsych.2014.05.024.

Kohane, Isaac S., Andrew McMurry, Griffin Weber, Douglas MacFadden, Leonard Rappaport, Louis Kunkel, Jonathan Bickel, et al. "The Co-Morbidity Burden of Children and Young Adults with Autism Spectrum Disorders." Edited by Neil R. Smalheiser. *PLoS ONE* 7, no. 4 (April 12, 2012): e33224. https://doi.org/10.1371/journal.pone.0033224.

Kojima, Masaki, Walid Yassin, Keiho Owada, Yuta Aoki, Hitoshi Kuwabara, Tatsunobu Natsubori, Norichika Iwashiro, et al. "Neuroanatomical Correlates of Advanced Paternal and Maternal Age at Birth in Autism Spectrum Disorder." *Cerebral Cortex (New York,*

N.Y.: 1991) 29, no. 6 (June 1, 2019): 2524–32. https://doi.org/10.1093/cercor/bhy122.

———. "Neuroanatomical Correlates of Advanced Paternal and Maternal Age at Birth in Autism Spectrum Disorder." *Cerebral Cortex* 29, no. 6 (June 1, 2019): 2524–32. https://doi.org/10.1093/cercor/bhy122.

Kolevzon, Alexander, Jeffrey H. Newcorn, Lauren Kryzak, William Chaplin, Dryden Watner, Eric Hollander, Christopher J. Smith, Edwin H. Cook, and Jeremy M. Silverman. "Relationship between Whole Blood Serotonin and Repetitive Behaviors in Autism." *Psychiatry Research* 175, no. 3 (February 2010): 274–76. https://doi.org/10.1016/j.psychres.2009.02.008.

Kolevzon, et al. "Selective Serotonin Reuptake Inhibitors in Autism: A Review of Efficacy and Tolerability," 2006.

Kõlves, Kairi, Cecilie Fitzgerald, Merete Nordentoft, Stephen James Wood, and Annette Erlangsen. "Assessment of Suicidal Behaviors Among Individuals With Autism Spectrum Disorder in Denmark." *JAMA Network Open* 4, no. 1 (January 12, 2021): e2033565. https://doi.org/10.1001/jamanetworkopen.2020.33565.

Kommu, John Vijay Sagar, Gayathri K.R., Shoba Srinath, Satish Chandra Girimaji, Shekhar P. Seshadri, Gururaj Gopalakrishna, and Subbakrishna Doddaballapura K. "Profile of Two Hundred Children with Autism Spectrum Disorder from a Tertiary Child and Adolescent Psychiatry Centre." *Asian Journal of Psychiatry* 28 (August 2017): 51–56. https://doi.org/10.1016/j.ajp.2017.03.017.

Krenzelok, Edward P., and Mike A. Royal. "Confusion." *Drugs in R&D* 12, no. 2 (June 2012): 45–48. https://doi.org/10.2165/11633010-000000000-00000.

Kriebel, David. "How Much Evidence Is Enough? Conventions of Causal Inference." *LAW AND CONTEMPORARY PROBLEMS* 72 (n.d.).

Kumar, Geetha, and Robert A. Steer. "Factorial Validity of the Conners' Parent Rating Scale-Revised: Short Form With Psychiatric Outpatients." *Journal of Personality Assessment* 80, no. 3 (June 2003): 252–59. https://doi.org/10.1207/S15327752JPA8003_04.

Kunreuther, Elizabeth. "Autism Spectrum Disorder and Substance Use Disorder." *Child and Adolescent Psychiatric Clinics of North America* 29, no. 3 (July 2020): 467–81. https://doi.org/10.1016/j.chc.2020.03.002.

Kuriakose, Sarah, Beryl Filton, Mollie Marr, Eugene Okparaeke, Paige Cervantes, Matthew Siegel, Sarah Horwitz, and Jennifer Havens. "Does an Autism Spectrum Disorder Care Pathway Improve Care for Children and Adolescents with ASD in Inpatient Psychiatric Units?" *Journal of Autism and Developmental Disorders* 48, no. 12 (December 2018): 4082–89. https://doi.org/10.1007/s10803-018-3666-y.

Kushima, Itaru, Branko Aleksic, Masahiro Nakatochi, Teppei Shimamura, Takashi Okada, Yota Uno, Mako Morikawa, et al. "Comparative Analyses of Copy-Number Variation in Autism Spectrum Disorder and Schizophrenia Reveal Etiological Overlap and Biological Insights." *Cell Reports* 24, no. 11 (September 2018): 2838–56. https://doi.org/10.1016/j.celrep.2018.08.022.

Kushki, Azadeh, Evdokia Anagnostou, Christopher Hammill, Pierre Duez, Jessica Brian, Alana Iaboni, Russell Schachar, Jennifer Crosbie, Paul Arnold, and Jason P. Lerch. "Examining Overlap and Homogeneity in ASD, ADHD, and OCD: A Data-Driven, Diagnosis-Agnostic Approach." *Translational Psychiatry* 9 (November 26, 2019): 318. https://doi.org/10.1038/s41398-019-0631-2.

Kushki, Azadeh, Robyn E Cardy, Sina Panahandeh, Mahan Malihi, Christopher Hammill, Jessica Brian, Alana Iaboni, et al. "Cross-Diagnosis Structural Correlates of Autistic-Like Social Communication Differences." *Cerebral Cortex (New York, NY)* 31, no. 11 (June 3, 2021): 5067–76. https://doi.org/10.1093/cercor/bhab142.

Labba, Nils-Anders, Hallvard Austin Wæhler, Nora Houdaifi, Denis Zosen, Fred Haugen, Ragnhild Elisabeth Paulsen, Mussie Ghezu Hadera, and Ragnhild Eskeland. "Paracetamol Perturbs Neuronal Arborization and Disrupts the Cytoskeletal Proteins SPTBN1 and TUBB3 in Both Human and Chicken in Vitro Models." *Toxicology and Applied Pharmacology* 449 (August 2022): 116130. https://doi.org/10.1016/j.taap.2022.116130.

Lafenêtre, Pauline, Francis Chaouloff, and Giovanni Marsicano. "Bidirectional Regulation of Novelty-Induced Behavioral Inhibition by the Endocannabinoid System." *Neuropharmacology*, Attention deficit hyperactivity disorder (ADHD): Improved understanding and novel drug treatment, 57, no. 7 (December 1, 2009): 715–21. https://doi.org/10.1016/j.neuropharm.2009.07.014.

Lai, Meng-Chuan, Caroline Kassee, Richard Besney, Sarah Bonato, Laura Hull, William Mandy, Peter Szatmari, and Stephanie H Ameis. "Prevalence of Co-Occurring Mental Health Diagnoses in the Autism Population: A Systematic Review and Meta-Analysis." *The Lancet Psychiatry* 6, no. 10 (October 2019): 819–29. https://doi.org/10.1016/S2215-0366(19)30289-5.

Lam, Kristen S. L., and Michael G. Aman. "The Repetitive Behavior Scale-Revised: Independent Validation in Individuals with Autism Spectrum Disorders." *Journal of Autism and Developmental Disorders* 37, no. 5 (May 2007): 855–66. https://doi.org/10.1007/s10803-006-0213-z.

Lam, Kristen S.L., James W. Bodfish, and Joseph Piven. "Evidence for Three Subtypes of Repetitive Behavior in Autism That Differ in Familiality and Association with Other Symptoms: Evidence for Three Subtypes of Repetitive Behavior in Autism." *Journal of Child Psychology and Psychiatry* 49, no. 11 (October 27, 2008): 1193–1200. https://doi.org/10.1111/j.1469-7610.2008.01944.x.

Larsson, Heidi Jeanet, William W. Eaton, Kreesten Meldgaard Madsen, Mogens Vestergaard, Anne Vingaard Olesen, Esben Agerbo, Diana Schendel, Poul Thorsen, and Preben Bo Mortensen. "Risk Factors for Autism: Perinatal Factors, Parental Psychiatric History, and Socioeconomic Status." *American Journal of Epidemiology* 161, no. 10 (May 15, 2005): 916–25. https://doi.org/10.1093/aje/kwi123.

Laue, Hannah E, Raphael Cassoulet, Nadia Abdelouahab, Yasmine K Serme-Gbedo, Anne-Sandrine Desautels, Kasey J M Brennan, Jean-Philippe Bellenger, et al. "Association Between Meconium Acetaminophen and Childhood Neurocognitive Development in

GESTE, a Canadian Cohort Study." *Toxicological Sciences* 167, no. 1 (January 1, 2019): 138–44. https://doi.org/10.1093/toxsci/kfy222.

Laue, Hannah E., Raphael Cassoulet, Jean-Philippe Bellenger, Andrea A. Baccarelli, and Larissa Takser. "Meconium: A Novel Biomarker of In Utero Exposure to Acetaminophen and Caffeine." *ISEE Conference Abstracts*, February 1, 2018. https://doi.org/10.1289/isee.2017.2017-848.

Laue, Hannah E., Yike Shen, Tessa R. Bloomquist, Haotian Wu, Kasey J. M. Brennan, Raphael Cassoulet, Erin Wilkie, et al. "In Utero Exposure to Caffeine and Acetaminophen, the Gut Microbiome, and Neurodevelopmental Outcomes: A Prospective Birth Cohort Study." *International Journal of Environmental Research and Public Health* 19, no. 15 (July 30, 2022): 9357. https://doi.org/10.3390/ijerph19159357.

Lau-Zhu, Alex, Anne Fritz, and Gráinne McLoughlin. "Overlaps and Distinctions between Attention Deficit/Hyperactivity Disorder and Autism Spectrum Disorder in Young Adulthood: Systematic Review and Guiding Framework for EEG-Imaging Research." *Neuroscience and Biobehavioral Reviews* 96 (January 2019): 93–115. https://doi.org/10.1016/j.neubiorev.2018.10.009.

Lebwohl, Benjamin, Linnea Haggård, Louise Emilsson, Jonas Söderling, Bjorn Roelstraete, Agnieszka Butwicka, Peter H. R. Green, and Jonas F. Ludvigsson. "Psychiatric Disorders in Patients With a Diagnosis of Celiac Disease During Childhood From 1973 to 2016." *Clinical Gastroenterology and Hepatology: The Official Clinical Practice Journal of the American Gastroenterological Association* 19, no. 10 (October 2021): 2093-2101.e13. https://doi.org/10.1016/j.cgh.2020.08.018.

Lecavalier, Luc. "Behavioral and Emotional Problems in Young People with Pervasive Developmental Disorders: Relative Prevalence, Effects of Subject Characteristics, and Empirical Classification." *Journal of Autism and Developmental Disorders* 36, no. 8 (December 14, 2006): 1101–14. https://doi.org/10.1007/s10803-006-0147-5.

Lecavalier, Luc, James Bodfish, Clare Harrop, Allison Whitten, Desiree Jones, Jill Pritchett, Richard Faldowski, and Brian Boyd. "Development of the Behavioral Inflexibility Scale for Children with Autism Spectrum Disorder and Other Developmental Disabilities." *Autism Research* 13, no. 3 (March 2020): 489–99. https://doi.org/10.1002/aur.2257.

Lee, Jin Kyung, Koji Abe, Arlene S. Bridges, Nita J. Patel, Thomas J. Raub, Gary M. Pollack, and Kim L. R. Brouwer. "Sex-Dependent Disposition of Acetaminophen Sulfate and Glucuronide in the in Situ Perfused Mouse Liver." *Drug Metabolism and Disposition* 37, no. 9 (September 2009): 1916–21. https://doi.org/10.1124/dmd.109.026815.

Lee, Ryan H, Elizabeth A Mills, Neil Schwartz, Mark R Bell, Katherine E Deeg, Edward S Ruthazer, Nicholas Marsh-Armstrong, and Carlos D Aizenman. "Neurodevelopmental Effects of Chronic Exposure to Elevated Levels of Pro-Inflammatory Cytokines in a Developing Visual System." *Neural Development* 5, no. 1 (December 2010): 2. https://doi.org/10.1186/1749-8104-5-2.

Lee, Tsung-Min, Kuan-Min Lee, Chuan-Ya Lee, Hsin-Chien Lee, Ka-Wai Tam, and El-Wui Loh. “Effectiveness of *N* -Acetylcysteine in Autism Spectrum Disorders: A Meta-Analysis of Randomized Controlled Trials.” *Australian & New Zealand Journal of Psychiatry* 55, no. 2 (February 2021): 196–206. https://doi.org/10.1177/0004867420952540.

Leekam, Susan, Jonathan Tandos, Helen McConachie, Elizabeth Meins, Kathryn Parkinson, Charlotte Wright, Michelle Turner, Bronia Arnott, Lucia Vittorini, and Ann Le Couteur. “Repetitive Behaviours in Typically Developing 2-Year-Olds.” *Journal of Child Psychology and Psychiatry* 48, no. 11 (November 2007): 1131–38. https://doi.org/10.1111/j.1469-7610.2007.01778.x.

Leung, Rachel C., and Konstantine K. Zakzanis. “Brief Report: Cognitive Flexibility in Autism Spectrum Disorders: A Quantitative Review.” *Journal of Autism and Developmental Disorders* 44, no. 10 (October 2014): 2628–45. https://doi.org/10.1007/s10803-014-2136-4.

Li, Chen, Peter M. Jones, and Shanta J. Persaud. “Role of the Endocannabinoid System in Food Intake, Energy Homeostasis and Regulation of the Endocrine Pancreas.” *Pharmacology & Therapeutics* 129, no. 3 (March 2011): 307–20. https://doi.org/10.1016/j.pharmthera.2010.10.006.

Li, Cheng, Yongqi Bai, Chuan Jin, Fangfang Zhong, Qulian Guo, and Wenjun Liu. “Efficacy and Safety of Fluoxetine in Autism Spectrum Disorder: A Meta-Analysis.” *American Journal of Therapeutics* 27, no. 3 (May 2020): e312–15. https://doi.org/10.1097/MJT.0000000000000978.

Liew, Zeyan, Cathrine Carlsen Bach, Robert F. Asarnow, Beate Ritz, and Jørn Olsen. “Paracetamol Use during Pregnancy and Attention and Executive Function in Offspring at Age 5 Years.” *International Journal of Epidemiology* 45, no. 6 (December 28, 2016): dyw296. https://doi.org/10.1093/ije/dyw296.

Liew, Zeyan, Marianthi-Anna Kioumourtzoglou, Andrea L Roberts, Éilis J O’Reilly, Alberto Ascherio, and Marc G Weisskopf. “Use of Negative Control Exposure Analysis to Evaluate Confounding: An Example of Acetaminophen Exposure and Attention-Deficit/Hyperactivity Disorder in Nurses’ Health Study II.” *American Journal of Epidemiology* 188, no. 4 (April 1, 2019): 768–75. https://doi.org/10.1093/aje/kwy288.

Liew, Zeyan, Beate Ritz, and Jørn Olsen. “Characteristics of Acetaminophen Users Compared With Nonusers During Pregnancy, Behavioral Problems, and Hyperkinetic Disorders—Reply.” *JAMA Pediatrics* 168, no. 9 (September 1, 2014): 865. https://doi.org/10.1001/jamapediatrics.2014.983.

Liew, Zeyan, Beate Ritz, Cristina Rebordosa, Pei-Chen Lee, and Jørn Olsen. “Acetaminophen Use During Pregnancy, Behavioral Problems, and Hyperkinetic Disorders.” *JAMA Pediatrics* 168, no. 4 (2014): 8. https://doi.org/doi:10.1001/jamapediatrics.2013.4914.

Liew, Zeyan, Beate Ritz, Jasveer Virk, Onyebuchi A. Arah, and Jørn Olsen. “Prenatal Use of Acetaminophen and Child IQ: A Danish Cohort Study.” *Epidemiology* 27, no. 6 (November 2016): 912–18. https://doi.org/10.1097/EDE.0000000000000540.

Liew, Zeyan, Beate Ritz, Jasveer Virk, and Jørn Olsen. "Maternal Use of Acetaminophen during Pregnancy and Risk of Autism Spectrum Disorders in Childhood: A Danish National Birth Cohort Study." *Autism Research: Official Journal of the International Society for Autism Research* 9, no. 9 (September 2016): 951–58. https://doi.org/10.1002/aur.1591.

Likhitweerawong, Narueporn, Chanisa Thonusin, Nonglak Boonchooduang, Orawan Louthrenoo, Intawat Nookaew, Nipon Chattipakorn, and Siriporn C. Chattipakorn. "Profiles of Urine and Blood Metabolomics in Autism Spectrum Disorders." *Metabolic Brain Disease* 36, no. 7 (October 2021): 1641–71. https://doi.org/10.1007/s11011-021-00788-3.

Lin, C. Enjey, and Robert Koegel. "Treatment for Higher-Order Restricted Repetitive Behaviors (H-RRB) in Children with Autism Spectrum Disorder." *Journal of Autism and Developmental Disorders* 48, no. 11 (November 2018): 3831–45. https://doi.org/10.1007/s10803-018-3637-3.

Lin, Haixi, Qingxia Lin, Hailong Li, Meihao Wang, Hong Chen, Yan Liang, Xuan Bu, et al. "Functional Connectivity of Attention-Related Networks in Drug-Naïve Children With ADHD." *Journal of Attention Disorders* 25, no. 3 (February 2021): 377–88. https://doi.org/10.1177/1087054718802017.

Lin, Mingyan, Dejian Zhao, Anastasia Hrabovsky, Erika Pedrosa, Deyou Zheng, and Herbert M. Lachman. "Heat Shock Alters the Expression of Schizophrenia and Autism Candidate Genes in an Induced Pluripotent Stem Cell Model of the Human Telencephalon." Edited by Sandy D. Westerheide. *PLoS ONE* 9, no. 4 (April 15, 2014): e94968. https://doi.org/10.1371/journal.pone.0094968.

Lin, Yi-Tsen, Yang-Ching Chen, Susan Shur-Fen Gau, Te-Huei Yeh, Hsien-Yu Fan, Yu-Ya Hwang, and Yungling Leo Lee. "Associations between Allergic Diseases and Attention Deficit Hyperactivity/Oppositional Defiant Disorders in Children." *Pediatric Research* 80, no. 4 (October 2016): 480–85. https://doi.org/10.1038/pr.2016.111.

Liu, De-Yi, Xue-Mei Shen, Fang-Fen Yuan, Ou-Yang Guo, Yan Zhong, Jian-Guo Chen, Ling-Qiang Zhu, and Jing Wu. "The Physiology of BDNF and Its Relationship with ADHD." *Molecular Neurobiology* 52, no. 3 (December 1, 2015): 1467–76. https://doi.org/10.1007/s12035-014-8956-6.

Liu, Xian, Mingyang Zou, Caihong Sun, Lijie Wu, and Wen-Xiong Chen. "Prenatal Folic Acid Supplements and Offspring's Autism Spectrum Disorder: A Meta-Analysis and Meta-Regression." *Journal of Autism and Developmental Disorders* 52, no. 2 (February 2022): 522–39. https://doi.org/10.1007/s10803-021-04951-8.

Liu, Xiaoqin, Esben Agerbo, Katja G Ingstrup, Katherine Musliner, Samantha Meltzer-Brody, Veerle Bergink, and Trine Munk-Olsen. "Antidepressant Use during Pregnancy and Psychiatric Disorders in Offspring: Danish Nationwide Register Based Cohort Study." *BMJ*, September 6, 2017, j3668. https://doi.org/10.1136/bmj.j3668.

Liu, Xiaoqin, Søren Dalsgaard, Trine-Munk Olsen, Jiong Li, Rosalind J. Wright, and Natalie Momen. "Parental Asthma Occurrence, Exacerbations and Risk of Attention-Deficit/Hyperactivity Disorder." *Brain, Behavior, and Immunity* 82 (November 2019): 302–8. https://doi.org/10.1016/j.bbi.2019.08.198.

Liu, Xukun, Jing Lin, Huajie Zhang, Naseer Ullah Khan, Jun Zhang, Xiaoxiao Tang, Xueshan Cao, and Liming Shen. "Oxidative Stress in Autism Spectrum Disorder—Current Progress of Mechanisms and Biomarkers." *Frontiers in Psychiatry* 13 (March 1, 2022): 813304. https://doi.org/10.3389/fpsyt.2022.813304.

Loebel, Antony, Matthew Brams, Robert S. Goldman, Robert Silva, David Hernandez, Ling Deng, Raymond Mankoski, and Robert L. Findling. "Lurasidone for the Treatment of Irritability Associated with Autistic Disorder." *Journal of Autism and Developmental Disorders* 46, no. 4 (April 2016): 1153–63. https://doi.org/10.1007/s10803-015-2628-x.

Loftin, Rachel L., Samuel L. Odom, and Johanna F. Lantz. "Social Interaction and Repetitive Motor Behaviors." *Journal of Autism and Developmental Disorders* 38, no. 6 (July 2008): 1124–35. https://doi.org/10.1007/s10803-007-0499-5.

Loke, Yuk Jing, Anthony John Hannan, and Jeffrey Mark Craig. "The Role of Epigenetic Change in Autism Spectrum Disorders." *Frontiers in Neurology* 6 (May 26, 2015): 107. https://doi.org/10.3389/fneur.2015.00107.

Long, Manhai, Mandana Ghisari, Lisbeth Kjeldsen, Maria Wielsøe, Bent Nørgaard-Pedersen, Erik Lykke Mortensen, Morsi W. Abdallah, and Eva C. Bonefeld-Jørgensen. "Autism Spectrum Disorders, Endocrine Disrupting Compounds, and Heavy Metals in Amniotic Fluid: A Case-Control Study." *Molecular Autism* 10 (2019): 1. https://doi.org/10.1186/s13229-018-0253-1.

Lord, Catherine, Mayada Elsabbagh, Gillian Baird, and Jeremy Veenstra-Vanderweele. "Autism Spectrum Disorder." *The Lancet* 392, no. 10146 (August 2018): 508–20. https://doi.org/10.1016/S0140-6736(18)31129-2.

Loyer Carbonneau, Maryanne, Martin Demers, Marc Bigras, and Marie-Claude Guay. "Meta-Analysis of Sex Differences in ADHD Symptoms and Associated Cognitive Deficits." *Journal of Attention Disorders* 25, no. 12 (October 2021): 1640–56. https://doi.org/10.1177/1087054720923736.

Lugo, Jorge, Christian Fadeuilhe, Laura Gisbert, Imanol Setien, Mercedes Delgado, Montserrat Corrales, Vanesa Richarte, and Josep Antoni Ramos-Quiroga. "Sleep in Adults with Autism Spectrum Disorder and Attention Deficit/Hyperactivity Disorder: A Systematic Review and Meta-Analysis." *European Neuropsychopharmacology: The Journal of the European College of Neuropsychopharmacology* 38 (September 2020): 1–24. https://doi.org/10.1016/j.euroneuro.2020.07.004.

Lukito, Steve, Luke Norman, Christina Carlisi, Joaquim Radua, Heledd Hart, Emily Simonoff, and Katya Rubia. "Comparative Meta-Analyses of Brain Structural and Functional Abnormalities during Cognitive Control in Attention-Deficit/Hyperactivity Disorder and Autism Spectrum Disorder." *Psychological Medicine* 50, no. 6 (April 2020): 894–919. https://doi.org/10.1017/S0033291720000574.

Luna, Ruth Ann, Numan Oezguen, Miriam Balderas, Alamelu Venkatachalam, Jessica K. Runge, James Versalovic, Jeremy Veenstra-VanderWeele, George M. Anderson, Tor Savidge, and Kent C. Williams. "Distinct Microbiome-Neuroimmune Signatures Correlate With Functional Abdominal Pain in Children With Autism Spectrum Disorder." *Cellular and Molecular Gastroenterology and Hepatology* 3, no. 2 (March 2017): 218–30. https://doi.org/10.1016/j.jcmgh.2016.11.008.

Luteijn, E F, M Althaus, S Jackson, and F Volkmar. "A Study of Social Problems in Children with PDD-NOS and ADHD." *Adolescent Psychiatry* 9, no. 3 (2000).

Lyall, Kristen, Paul Ashwood, Judy Van de Water, and I. Hertz-Picciotto. "Maternal Immune-Mediated Conditions, Autism Spectrum Disorders, and Developmental Delay." *Journal of Autism and Developmental Disorders* 44, no. 7 (July 2014): 1546–55. https://doi.org/10.1007/s10803-013-2017-2.

Lyall, Kristen, Judy Van De Water, Paul Ashwood, and Irva Hertz-Picciotto. "Asthma and Allergies in Children With Autism Spectrum Disorders: Results From the CHARGE Study: Asthma and Allergies in Autism." *Autism Research* 8, no. 5 (October 2015): 567–74. https://doi.org/10.1002/aur.1471.

Lytle, Sarah, Andrew Hunt, Sonal Moratschek, Marcie Hall-Mennes, and Martha Sajatovic. "Youth With Autism Spectrum Disorder in the Emergency Department." *The Journal of Clinical Psychiatry* 79, no. 3 (June 27, 2018). https://doi.org/10.4088/JCP.17r11506.

Maenner, Matthew J., Kelly A. Shaw, Amanda V. Bakian, Deborah A. Bilder, Maureen S. Durkin, Amy Esler, Sarah M. Furnier, et al. "Prevalence and Characteristics of Autism Spectrum Disorder Among Children Aged 8 Years - Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2018." *Morbidity and Mortality Weekly Report. Surveillance Summaries (Washington, D.C.: 2002)* 70, no. 11 (December 3, 2021): 1–16. https://doi.org/10.15585/mmwr.ss7011a1.

Maenner, Matthew J, Zachary Warren, Ashley Robinson Williams, Esther Amoakohene, Deborah A Bilder, Maureen S Durkin, Robert T Fitzgerald, et al. "Prevalence and Characteristics of Autism Spectrum Disorder Among Children Aged 8 Years — Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2020," 2020.

———. "Prevalence and Characteristics of Autism Spectrum Disorder Among Children Aged 8 Years — Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2020," 2023.

Mahendiran, Tania, Annie Dupuis, Jennifer Crosbie, Stelios Georgiades, Elizabeth Kelley, Xudong Liu, Robert Nicolson, Russell Schachar, Evdokia Anagnostou, and Jessica Brian. "Sex Differences in Social Adaptive Function in Autism Spectrum Disorder and Attention-Deficit Hyperactivity Disorder." *Frontiers in Psychiatry* 10 (September 12, 2019): 607. https://doi.org/10.3389/fpsyt.2019.00607.

Maimburg, R. D., and M. Væth. "Perinatal Risk Factors and Infantile Autism." *Acta Psychiatrica Scandinavica* 114, no. 4 (2006): 257–64. https://doi.org/10.1111/j.1600-0447.2006.00805.x.

Malkova, Natalia V., Collin Z. Yu, Elaine Y. Hsiao, Marlyn J. Moore, and Paul H. Patterson. "Maternal Immune Activation Yields Offspring Displaying Mouse Versions of the Three Core Symptoms of Autism." *Brain, Behavior, and Immunity* 26, no. 4 (May 2012): 607–16. https://doi.org/10.1016/j.bbi.2012.01.011.

Mandal, Mili, Atara C. Marzouk, Robert Donnelly, and Nicholas M. Ponzio. "Maternal Immune Stimulation during Pregnancy Affects Adaptive Immunity in Offspring to Promote Development of TH17 Cells." *Brain, Behavior, and Immunity* 25, no. 5 (July 2011): 863–71. https://doi.org/10.1016/j.bbi.2010.09.011.

Manivasagam, Thamilarasan, Selvaraj Arunadevi, Mustafa Mohamed Essa, Chidambaram SaravanaBabu, Anupom Borah, Arokiasamy Justin Thenmozhi, and M. Walid Qoronfleh. "Role of Oxidative Stress and Antioxidants in Autism." *Advances in Neurobiology* 24 (2020): 193–206. https://doi.org/10.1007/978-3-030-30402-7_7.

Mann, Joshua R., Suzanne McDermott, Haikun Bao, James Hardin, and Anthony Gregg. "Pre-Eclampsia, Birth Weight, and Autism Spectrum Disorders." *Journal of Autism and Developmental Disorders* 40, no. 5 (May 2010): 548–54. https://doi.org/10.1007/s10803-009-0903-4.

Manzardo, A.M., R. Henkhaus, S. Dhillon, and M.G. Butler. "Plasma Cytokine Levels in Children with Autistic Disorder and Unrelated Siblings." *International Journal of Developmental Neuroscience* 30, no. 2 (April 2012): 121–27. https://doi.org/10.1016/j.ijdevneu.2011.12.003.

Marcus, Ronald N., Randall Owen, Lisa Kamen, George Manos, Robert D. McQuade, William H. Carson, and Michael G. Aman. "A Placebo-Controlled, Fixed-Dose Study of Aripiprazole in Children and Adolescents With Irritability Associated With Autistic Disorder." *Journal of the American Academy of Child & Adolescent Psychiatry* 48, no. 11 (November 2009): 1110–19. https://doi.org/10.1097/CHI.0b013e3181b76658.

Marí-Bauset, Salvador, Carolina Donat-Vargas, Agustín Llópis-González, Amelia Marí-Sanchis, Isabel Peraita-Costa, Juan Llopis-Morales, and María Morales-Suárez-Varela. "Endocrine Disruptors and Autism Spectrum Disorder in Pregnancy: A Review and Evaluation of the Quality of the Epidemiological Evidence." *Children* 5, no. 12 (November 23, 2018): 157. https://doi.org/10.3390/children5120157.

Marrus, Natasha, Jeremy Veenstra-VanderWeele, Jessica A Hellings, Kimberly A Stigler, Ludwik Szymanski, Bryan H King, L Lee Carlisle, Edwin H Cook, The American Academy of Child Adolescent Psychiatry (AACAP) Autism and Intellectual Disability Committee, and John R Pruett. "Training of Child and Adolescent Psychiatry Fellows in Autism and Intellectual Disability." *Autism* 18, no. 4 (May 2014): 471–75. https://doi.org/10.1177/1362361313477247.

Martin, Joanna, Marian L. Hamshere, Michael C. O'Donovan, Michael Rutter, and Anita Thapar. "Factor Structure of Autistic Traits in Children with ADHD." *Journal of Autism and Developmental Disorders* 44, no. 1 (January 2014): 204–15. https://doi.org/10.1007/s10803-013-1865-0.

Martinez-De La Torre, Adrian, Stefan Weiler, Dominic Stefan Bräm, Samuel S. Allemann, Hugo Kupferschmidt, and Andrea M. Burden. “National Poison Center Calls Before vs After Availability of High-Dose Acetaminophen (Paracetamol) Tablets in Switzerland.” *JAMA Network Open* 3, no. 10 (October 28, 2020): e2022897. https://doi.org/10.1001/jamanetworkopen.2020.22897.

Martínez-González, A. E., and J. A. Piqueras. “Validation of the Repetitive Behavior Scale-Revised in Spanish-Speakers Participants with Autism Spectrum Disorder.” *Journal of Autism and Developmental Disorders* 48, no. 1 (January 2018): 198–208. https://doi.org/10.1007/s10803-017-3276-0.

Marton, Imola, Judith Wiener, Maria Rogers, Chris Moore, and Rosemary Tannock. “Empathy and Social Perspective Taking in Children with Attention-Deficit/Hyperactivity Disorder.” *Journal of Abnormal Child Psychology* 37, no. 1 (January 2009): 107–18. https://doi.org/10.1007/s10802-008-9262-4.

Masarwa, Reem, Hagai Levine, Einat Gorelik, Shimon Reif, Amichai Perlman, and Ilan Matok. “Prenatal Exposure to Acetaminophen and Risk for Attention Deficit Hyperactivity Disorder and Autistic Spectrum Disorder: A Systematic Review, Meta-Analysis, and Meta-Regression Analysis of Cohort Studies.” *American Journal of Epidemiology* 187, no. 8 (August 1, 2018): 1817–27. https://doi.org/10.1093/aje/kwy086.

Masarwa, Reem, Robert W. Platt, and Kristian B. Filion. “Acetaminophen Use during Pregnancy and the Risk of Attention Deficit Hyperactivity Disorder: A Causal Association or Bias?” *Paediatric and Perinatal Epidemiology* 34, no. 3 (May 2020): 309–17. https://doi.org/10.1111/ppe.12615.

Masi, A, D S Quintana, N Glozier, A R Lloyd, I B Hickie, and A J Guastella. “Cytokine Aberrations in Autism Spectrum Disorder: A Systematic Review and Meta-Analysis.” *Molecular Psychiatry* 20, no. 4 (April 2015): 440–46. https://doi.org/10.1038/mp.2014.59.

Masi, Anne, Nicholas Glozier, Russell Dale, and Adam J. Guastella. “The Immune System, Cytokines, and Biomarkers in Autism Spectrum Disorder.” *Neuroscience Bulletin* 33, no. 2 (April 2017): 194–204. https://doi.org/10.1007/s12264-017-0103-8.

Masini, Elena, Eleonora Loi, Ana Florencia Vega-Benedetti, Marinella Carta, Giuseppe Doneddu, Roberta Fadda, and Patrizia Zavattari. “An Overview of the Main Genetic, Epigenetic and Environmental Factors Involved in Autism Spectrum Disorder Focusing on Synaptic Activity.” *International Journal of Molecular Sciences* 21, no. 21 (November 5, 2020): 8290. https://doi.org/10.3390/ijms21218290.

Massat, Isabelle, Hichem Slama, Thomas Villemonteix, Alison Mary, Simon Baijot, Ariadna Albajara Sáenz, Danièle Balériaux, Thierry Metens, Martin Kavec, and Philippe Peigneux. “Hyperactivity in Motor Response Inhibition Networks in Unmedicated Children with Attention Deficit-Hyperactivity Disorder.” *The World Journal of Biological Psychiatry* 19, no. 2 (February 17, 2018): 101–11. https://doi.org/10.1080/15622975.2016.1237040.

Mazefsky, Carla A., Ranita Anderson, Caitlin M. Conner, and Nancy Minshew. “Child Behavior Checklist Scores for School-Aged Children with Autism: Preliminary Evidence of Patterns Suggesting the Need for Referral.” *Journal of Psychopathology and Behavioral Assessment* 33, no. 1 (March 2011): 31–37. https://doi.org/10.1007/s10862-010-9198-1.

Mazefsky, Carla A., Diane L. Williams, and Nancy J. Minshew. “Variability in Adaptive Behavior in Autism: Evidence for the Importance of Family History.” *Journal of Abnormal Child Psychology* 36, no. 4 (May 2008): 591–99. https://doi.org/10.1007/s10802-007-9202-8.

McClain, Maryellen Brunson, Amber M. Hasty Mills, and Laura E. Murphy. “Inattention and Hyperactivity/Impulsivity among Children with Attention-Deficit/Hyperactivity-Disorder, Autism Spectrum Disorder, and Intellectual Disability.” *Research in Developmental Disabilities* 70 (November 2017): 175–84. https://doi.org/10.1016/j.ridd.2017.09.009.

McCue, Lena M., Louise H. Flick, Kimberly A. Twyman, Hong Xian, and Thomas E. Conturo. “Prevalence of Non-Febrile Seizures in Children with Idiopathic Autism Spectrum Disorder and Their Unaffected Siblings: A Retrospective Cohort Study.” *BMC Neurology* 16, no. 1 (December 2016): 245. https://doi.org/10.1186/s12883-016-0764-3.

McDougle, Christopher J., Samantha M. Landino, Arshya Vahabzadeh, Julia O’Rourke, Nicole R. Zurcher, Beate C. Finger, Michelle L. Palumbo, et al. “Toward an Immune-Mediated Subtype of Autism Spectrum Disorder.” *Brain Research* 1617 (August 2015): 72–92. https://doi.org/10.1016/j.brainres.2014.09.048.

McFadden, Kathryn, and Nancy J. Minshew. “Evidence for Dysregulation of Axonal Growth and Guidance in the Etiology of ASD.” *Frontiers in Human Neuroscience* 7 (2013): 671. https://doi.org/10.3389/fnhum.2013.00671.

McFayden, T. C., J. Albright, A. E. Muskett, and A. Scarpa. “Brief Report: Sex Differences in ASD Diagnosis—A Brief Report on Restricted Interests and Repetitive Behaviors.” *Journal of Autism and Developmental Disorders* 49, no. 4 (April 2019): 1693–99. https://doi.org/10.1007/s10803-018-3838-9.

McKinnon, Claire J., Adam T. Eggebrecht, Alexandre Todorov, Jason J. Wolff, Jed T. Elison, Chloe M. Adams, Abraham Z. Snyder, et al. “Restricted and Repetitive Behavior and Brain Functional Connectivity in Infants at Risk for Developing Autism Spectrum Disorder.” *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* 4, no. 1 (January 2019): 50–61. https://doi.org/10.1016/j.bpsc.2018.09.008.

McQuaid, Goldie A, Kevin A Pelphrey, Susan Y Bookheimer, Mirella Dapretto, Sara J Webb, Raphael A Bernier, James C McPartland, John D Van Horn, and Gregory L Wallace. “The Gap between IQ and Adaptive Functioning in Autism Spectrum Disorder: Disentangling Diagnostic and Sex Differences.” *Autism* 25, no. 6 (August 2021): 1565–79. https://doi.org/10.1177/1362361321995620.

Mechler, Konstantin, Tobias Banaschewski, Sarah Hohmann, and Alexander Häge. “Evidence-Based Pharmacological Treatment Options for ADHD in Children and Adolescents.” *Pharmacology & Therapeutics* 230 (February 2022): 107940.

https://doi.org/10.1016/j.pharmthera.2021.107940.

Meier, Sandra M, Liselotte Petersen, Diana E Schendel, Manuel Mattheisen, Preben B Mortensen, and Ole Mors. "Obsessive-Compulsive Disorder and Autism Spectrum Disorders: Longitudinal and Offspring Risk." Edited by Kenji Hashimoto. *PLOS ONE* 10, no. 11 (November 11, 2015): e0141703. https://doi.org/10.1371/journal.pone.0141703.

Melnyk, Stepan, George J. Fuchs, Eldon Schulz, Maya Lopez, Stephen G. Kahler, Jill J. Fussell, Jayne Bellando, et al. "Metabolic Imbalance Associated with Methylation Dysregulation and Oxidative Damage in Children with Autism." *Journal of Autism and Developmental Disorders* 42, no. 3 (March 2012): 367–77. https://doi.org/10.1007/s10803-011-1260-7.

Meltzer, Amory, and Judy Van De Water. "The Role of the Immune System in Autism Spectrum Disorder." *Neuropsychopharmacology* 42, no. 1 (January 2017): 284–98. https://doi.org/10.1038/npp.2016.158.

Meng, Wei-Dong, Shao-Jun Sun, Jie Yang, Rui-Xue Chu, Wenjun Tu, and Qiang Liu. "Elevated Serum Brain-Derived Neurotrophic Factor (BDNF) but Not BDNF Gene Val66Met Polymorphism Is Associated with Autism Spectrum Disorders." *Molecular Neurobiology* 54, no. 2 (March 2017): 1167–72. https://doi.org/10.1007/s12035-016-9721-9.

Meyer, Urs. "Prenatal Poly(I:C) Exposure and Other Developmental Immune Activation Models in Rodent Systems." *Biological Psychiatry* 75, no. 4 (February 2014): 307–15. https://doi.org/10.1016/j.biopsych.2013.07.011.

Michel, Maximilian, Martin J. Schmidt, and Karoly Mirnics. "Immune System Gene Dysregulation in Autism and Schizophrenia." *Developmental Neurobiology* 72, no. 10 (October 2012): 1277–87. https://doi.org/10.1002/dneu.22044.

Militerni, Roberto, Carmela Bravaccio, Carmelinda Falco, Cinzia Fico, and Mark T. Palermo. "Repetitive Behaviors in Autistic Disorder." *European Child & Adolescent Psychiatry* 11, no. 5 (October 2002): 210–18. https://doi.org/10.1007/s00787-002-0279-x.

Mill, Jonathan, and Arturas Petronis. "Pre- and Peri-Natal Environmental Risks for Attention-Deficit Hyperactivity Disorder (ADHD): The Potential Role of Epigenetic Processes in Mediating Susceptibility." *Journal of Child Psychology and Psychiatry* 49, no. 10 (2008): 1020–30. https://doi.org/10.1111/j.1469-7610.2008.01909.x.

Miller, V. M., Y. Zhu, C. Bucher, W. McGinnis, L. K. Ryan, A. Siegel, and S. Zalcman. "Gestational Flu Exposure Induces Changes in Neurochemicals, Affiliative Hormones and Brainstem Inflammation, in Addition to Autism-like Behaviors in Mice." *Brain, Behavior, and Immunity* 33 (October 2013): 153–63. https://doi.org/10.1016/j.bbi.2013.07.002.

Ming, Xue, T. Peter Stein, Virginia Barnes, Nelson Rhodes, and Lining Guo. "Metabolic Perturbance in Autism Spectrum Disorders: A Metabolomics Study." *Journal of Proteome Research* 11, no. 12 (December 7, 2012): 5856–62. https://doi.org/10.1021/pr300910n.

Mirabella, Giovanni. "Inhibitory Control and Impulsive Responses in Neurodevelopmental Disorders." *Developmental Medicine & Child Neurology* 63, no. 5 (May 2021): 520–26. https://doi.org/10.1111/dmcn.14778.

Mirrasekhian, Elahe, Johan L. Å Nilsson, Kiseko Shionoya, Anders Blomgren, Peter M. Zygmunt, David Engblom, Edward D. Högestätt, and Anders Blomqvist. "The Antipyretic Effect of Paracetamol Occurs Independent of Transient Receptor Potential Ankyrin 1-Mediated Hypothermia and Is Associated with Prostaglandin Inhibition in the Brain." *FASEB Journal: Official Publication of the Federation of American Societies for Experimental Biology* 32, no. 10 (October 2018): 5751–59. https://doi.org/10.1096/fj.201800272R.

Missault, S., K. Van Den Eynde, W. Vanden Berghe, E. Fransen, A. Weeren, J.P. Timmermans, S. Kumar-Singh, and S. Dedeurwaerdere. "The Risk for Behavioural Deficits Is Determined by the Maternal Immune Response to Prenatal Immune Challenge in a Neurodevelopmental Model." *Brain, Behavior, and Immunity* 42 (November 2014): 138–46. https://doi.org/10.1016/j.bbi.2014.06.013.

Missig, Galen, Emery L Mokler, James O Robbins, Abigail J Alexander, Christopher J McDougle, and William A Carlezon. "Perinatal Immune Activation Produces Persistent Sleep Alterations and Epileptiform Activity in Male Mice." *Neuropsychopharmacology* 43, no. 3 (February 2018): 482–91. https://doi.org/10.1038/npp.2017.243.

Mitelman, Serge A., Marie-Cecile Bralet, M. Mehmet Haznedar, Eric Hollander, Lina Shihabuddin, Erin A. Hazlett, and Monte S. Buchsbaum. "Diametrical Relationship between Gray and White Matter Volumes in Autism Spectrum Disorder and Schizophrenia." *Brain Imaging and Behavior* 11, no. 6 (December 2017): 1823–35. https://doi.org/10.1007/s11682-016-9648-9.

Mitelman, Serge A., Marie-Cecile Bralet, M. Mehmet Haznedar, Eric Hollander, Lina Shihabuddin, Erin A. Hazlett, and Monte S. Buchsbaum. "Positron Emission Tomography Assessment of Cerebral Glucose Metabolic Rates in Autism Spectrum Disorder and Schizophrenia." *Brain Imaging and Behavior* 12, no. 2 (April 2018): 532–46. https://doi.org/10.1007/s11682-017-9721-z.

Mitelman, Serge A., Monte S. Buchsbaum, Derek S. Young, M. Mehmet Haznedar, Eric Hollander, Lina Shihabuddin, Erin A. Hazlett, and Marie-Cecile Bralet. "Increased White Matter Metabolic Rates in Autism Spectrum Disorder and Schizophrenia." *Brain Imaging and Behavior* 12, no. 5 (October 2018): 1290–1305. https://doi.org/10.1007/s11682-017-9785-9.

Miyasaka, Mami, Shogo Kajimura, and Michio Nomura. "Biases in Understanding Attention Deficit Hyperactivity Disorder and Autism Spectrum Disorder in Japan." *Frontiers in Psychology* 9 (February 28, 2018): 244. https://doi.org/10.3389/fpsyg.2018.00244.

Miyazaki, Celine, Momoko Koyama, Erika Ota, Toshiyuki Swa, Linda B. Mlunde, Rachel M. Amiya, Yoshiyuki Tachibana, Kiwako Yamamoto-Hanada, and Rintaro Mori. "Allergic Diseases in Children with Attention Deficit Hyperactivity Disorder: A Systematic Review and Meta-Analysis." *BMC Psychiatry* 17 (March 31, 2017): 120. https://doi.org/10.1186/s12888-017-1281-7.

Moffitt, Terrie E., Louise Arseneault, Daniel Belsky, Nigel Dickson, Robert J. Hancox, HonaLee Harrington, Renate Houts, et al. “A Gradient of Childhood Self-Control Predicts Health, Wealth, and Public Safety.” *Proceedings of the National Academy of Sciences of the United States of America* 108, no. 7 (February 15, 2011): 2693–98. https://doi.org/10.1073/pnas.1010076108.

Molloy, Cynthia A., Donna S. Murray, Rachel Akers, Terry Mitchell, and Patricia Manning-Courtney. “Use of the Autism Diagnostic Observation Schedule (ADOS) in a Clinical Setting.” *Autism* 15, no. 2 (March 2011): 143–62. https://doi.org/10.1177/1362361310379241.

Moore, R. A., S. Derry, P. J. Wiffen, S. Straube, and D. J. Aldington. “Overview Review: Comparative Efficacy of Oral Ibuprofen and Paracetamol (Acetaminophen) across Acute and Chronic Pain Conditions.” *European Journal of Pain (London, England)* 19, no. 9 (October 2015): 1213–23. https://doi.org/10.1002/ejp.649.

Morett, Laura M., Kirsten O’Hearn, Beatriz Luna, and Avniel Singh Ghuman. “Altered Gesture and Speech Production in ASD Detract from In-Person Communicative Quality.” *Journal of Autism and Developmental Disorders* 46, no. 3 (March 2016): 998–1012. https://doi.org/10.1007/s10803-015-2645-9.

Morgan, John T., Gursharan Chana, Ian Abramson, Katerina Semendeferi, Eric Courchesne, and Ian P. Everall. “Abnormal Microglial–Neuronal Spatial Organization in the Dorsolateral Prefrontal Cortex in Autism.” *Brain Research* 1456 (May 2012): 72–81. https://doi.org/10.1016/j.brainres.2012.03.036.

Morgan, John T., Gursharan Chana, Carlos A. Pardo, Cristian Achim, Katerina Semendeferi, Jody Buckwalter, Eric Courchesne, and Ian P. Everall. “Microglial Activation and Increased Microglial Density Observed in the Dorsolateral Prefrontal Cortex in Autism.” *Biological Psychiatry* 68, no. 4 (August 2010): 368–76. https://doi.org/10.1016/j.biopsych.2010.05.024.

Moriuchi, Jennifer M., Ami Klin, and Warren Jones. “Mechanisms of Diminished Attention to Eyes in Autism.” *American Journal of Psychiatry* 174, no. 1 (January 2017): 26–35. https://doi.org/10.1176/appi.ajp.2016.15091222.

Morris-Rosendahl, Deborah J., and Marc-Antoine Crocq. “Neurodevelopmental Disorders—the History and Future of a Diagnostic Concept.” *Dialogues in Clinical Neuroscience* 22, no. 1 (March 2020): 65–72. https://doi.org/10.31887/DCNS.2020.22.1/macrocq.

Moster, Dag, Rolv Terje Lie, and Trond Markestad. “Long-Term Medical and Social Consequences of Preterm Birth.” *The New England Journal of Medicine* 359, no. 3 (July 17, 2008): 262–73. https://doi.org/10.1056/NEJMoa0706475.

Mostert-Kerckhoffs, Mandy A. L., Wouter G. Staal, Renske H. Houben, and Maretha V. De Jonge. “Stop and Change: Inhibition and Flexibility Skills Are Related to Repetitive Behavior in Children and Young Adults with Autism Spectrum Disorders.” *Journal of Autism and Developmental Disorders* 45, no. 10 (October 2015): 3148–58. https://doi.org/10.1007/s10803-015-2473-y.

Mouga, Susana, Cátia Café, Joana Almeida, Carla Marques, Frederico Duque, and Guiomar Oliveira. "Intellectual Profiles in the Autism Spectrum and Other Neurodevelopmental Disorders." *Journal of Autism and Developmental Disorders* 46, no. 9 (September 2016): 2940–55. https://doi.org/10.1007/s10803-016-2838-x.

Mukhopadhya, Indrani, Richard Hansen, Charlotte E. Nicholl, Yazeid A. Alhaidan, John M. Thomson, Susan H. Berry, Craig Pattinson, et al. "A Comprehensive Evaluation of Colonic Mucosal Isolates of Sutterella Wadsworthensis from Inflammatory Bowel Disease." Edited by Stefan Bereswill. *PLoS ONE* 6, no. 10 (October 31, 2011): e27076. https://doi.org/10.1371/journal.pone.0027076.

Mulligan, Aisling, Richard J. L. Anney, Myra O'Regan, Wai Chen, Louise Butler, Michael Fitzgerald, Jan Buitelaar, et al. "Autism Symptoms in Attention-Deficit/Hyperactivity Disorder: A Familial Trait Which Correlates with Conduct, Oppositional Defiant, Language and Motor Disorders." *Journal of Autism and Developmental Disorders* 39, no. 2 (February 2009): 197–209. https://doi.org/10.1007/s10803-008-0621-3.

"Multi-Health-Systems-Usd." Accessed June 1, 2023. https://storefront.mhs.com/collections/conners-cpt-3.

Murphy, Declan, Karen Glaser, Hannah Hayward, Hanna Eklund, Tim Cadman, James Findon, Emma Woodhouse, et al. "Improving Diagnosis of ASD in the Community: Tool Development." In *Crossing the Divide: A Longitudinal Study of Effective Treatments for People with Autism and Attention Deficit Hyperactivity Disorder across the Lifespan*. NIHR Journals Library, 2018. https://www.ncbi.nlm.nih.gov/books/NBK518664/.

Nader, Anne-Marie, Patricia Jelenic, and Isabelle Soulières. "Discrepancy between WISC-III and WISC-IV Cognitive Profile in Autism Spectrum: What Does It Reveal about Autistic Cognition?" Edited by Tiziana Zalla. *PLOS ONE* 10, no. 12 (December 16, 2015): e0144645. https://doi.org/10.1371/journal.pone.0144645.

Napolioni, Valerio, Benjamin Ober-Reynolds, Szabolcs Szelinger, Jason J Corneveaux, Traci Pawlowski, Sharman Ober-Reynolds, Janet Kirwan, et al. "Plasma Cytokine Profiling in Sibling Pairs Discordant for Autism Spectrum Disorder." *Journal of Neuroinflammation* 10, no. 1 (December 2013): 813. https://doi.org/10.1186/1742-2094-10-38.

Naranjo, C. A., U. Busto, E. M. Sellers, P. Sandor, I. Ruiz, E. A. Roberts, E. Janecek, C. Domecq, and D. J. Greenblatt. "A Method for Estimating the Probability of Adverse Drug Reactions." *Clinical Pharmacology and Therapeutics* 30, no. 2 (August 1981): 239–45. https://doi.org/10.1038/clpt.1981.154.

National Institute of Environmental Health Sciences. "The Infant Development and Environment Study (TIDES)." Accessed June 16, 2023. https://tools.niehs.nih.gov/cohorts/index.cfm/main/detail/search/true/ids/c109.

National Institute of Mental Health (NIMH). "About RDoC." Accessed May 11, 2023. https://www.nimh.nih.gov/research/research-funded-by-nimh/rdoc/about-rdoc.

Navarrete, Francisco, María Salud García-Gutiérrez, Rosa Jurado-Barba, Gabriel Rubio, Ani Gasparyan, Amaya Austrich-Olivares, and Jorge Manzanares. "Endocannabinoid System Components as Potential Biomarkers in Psychiatry." *Frontiers in Psychiatry* 11 (April

27, 2020): 315. https://doi.org/10.3389/fpsyt.2020.00315.

Naviaux, J C, M A Schuchbauer, K Li, L Wang, V B Risbrough, S B Powell, and R K Naviaux. "Reversal of Autism-like Behaviors and Metabolism in Adult Mice with Single-Dose Antipurinergic Therapy." *Translational Psychiatry* 4, no. 6 (June 17, 2014): e400–e400. https://doi.org/10.1038/tp.2014.33.

Naviaux, Robert K., Zarazuela Zolkipli, Lin Wang, Tomohiro Nakayama, Jane C. Naviaux, Thuy P. Le, Michael A. Schuchbauer, et al. "Antipurinergic Therapy Corrects the Autism-Like Features in the Poly(IC) Mouse Model." Edited by Madepalli K. Lakshmana. *PLoS ONE* 8, no. 3 (March 13, 2013): e57380. https://doi.org/10.1371/journal.pone.0057380.

Needham, Brittany D., Mark D. Adame, Gloria Serena, Destanie R. Rose, Gregory M. Preston, Mary C. Conrad, A. Stewart Campbell, et al. "Plasma and Fecal Metabolite Profiles in Autism Spectrum Disorder." *Biological Psychiatry* 89, no. 5 (March 1, 2021): 451–62. https://doi.org/10.1016/j.biopsych.2020.09.025.

Needham, Brittany D., Rima Kaddurah-Daouk, and Sarkis K. Mazmanian. "Gut Microbial Molecules in Behavioural and Neurodegenerative Conditions." *Nature Reviews. Neuroscience* 21, no. 12 (December 2020): 717–31. https://doi.org/10.1038/s41583-020-00381-0.

Nelson, Sacha B., and Vera Valakh. "Excitatory/Inhibitory Balance and Circuit Homeostasis in Autism Spectrum Disorders." *Neuron* 87, no. 4 (August 19, 2015): 684–98. https://doi.org/10.1016/j.neuron.2015.07.033.

Nestler, Eric J., Elizabeth Gould, Husseini Manji, Maja Buncan, Ronald S. Duman, Howard K. Greshenfeld, Ren Hen, et al. "Preclinical Models: Status of Basic Research in Depression." *Biological Psychiatry* 52, no. 6 (September 15, 2002): 503–28. https://doi.org/10.1016/s0006-3223(02)01405-1.

Neumann, I. D., G. Wegener, J. R. Homberg, H. Cohen, D. A. Slattery, J. Zohar, J. D. A. Olivier, and A. A. Mathé. "Animal Models of Depression and Anxiety: What Do They Tell Us about Human Condition?" *Progress in Neuro-Psychopharmacology & Biological Psychiatry* 35, no. 6 (August 1, 2011): 1357–75. https://doi.org/10.1016/j.pnpbp.2010.11.028.

Nezgovorova, V., C.J. Ferretti, B.P. Taylor, E. Shanahan, G. Uzunova, K. Hong, O. Devinsky, and E. Hollander. "Potential of Cannabinoids as Treatments for Autism Spectrum Disorders." *Journal of Psychiatric Research* 137 (May 2021): 194–201. https://doi.org/10.1016/j.jpsychires.2021.02.048.

Nichols, Helen M., Sarah Dababnah, Brittany Troen, Jessica Vezzoli, Rajneesh Mahajan, and Carla A. Mazefsky. "Racial Disparities in a Sample of Inpatient Youth with ASD." *Autism Research* 13, no. 4 (April 2020): 532–38. https://doi.org/10.1002/aur.2262.

Nickel, Kathrin, Simon Maier, Dominique Endres, Andreas Joos, Viktoria Maier, Ludger Tebartz Van Elst, and Almut Zeeck. "Systematic Review: Overlap Between Eating, Autism Spectrum, and Attention-Deficit/Hyperactivity Disorder." *Frontiers in Psychiatry* 10 (October 10, 2019): 708. https://doi.org/10.3389/fpsyt.2019.00708.

Nicolini, Chiara, and Margaret Fahnestock. "The Valproic Acid-Induced Rodent Model of Autism." *Experimental Neurology* 299 (January 2018): 217–27. https://doi.org/10.1016/j.expneurol.2017.04.017.

Nielsen, Philip Rising, Michael Eriksen Benros, and Søren Dalsgaard. "Associations Between Autoimmune Diseases and Attention-Deficit/Hyperactivity Disorder: A Nationwide Study." *Journal of the American Academy of Child & Adolescent Psychiatry* 56, no. 3 (March 1, 2017): 234-240.e1. https://doi.org/10.1016/j.jaac.2016.12.010.

Nigg, Joel T., and B. J. Casey. "An Integrative Theory of Attention-Deficit/ Hyperactivity Disorder Based on the Cognitive and Affective Neurosciences." *Development and Psychopathology* 17, no. 3 (2005): 785–806. https://doi.org/10.1017/S0954579405050376.

Nijmeijer, J. S., P. J. Hoekstra, R. B. Minderaa, J. K. Buitelaar, M. E. Altink, C. J. M. Buschgens, E. A. Fliers, N. N. J. Rommelse, J. A. Sergeant, and C. A. Hartman. "PDD Symptoms in ADHD, an Independent Familial Trait?" *Journal of Abnormal Child Psychology* 37, no. 3 (April 2009): 443–53. https://doi.org/10.1007/s10802-008-9282-0.

Nitsche, Joshua F., Avinash S. Patil, Loralie J. Langman, Hannah J. Penn, Douglas Derleth, William J. Watson, and Brian C. Brost. "Transplacental Passage of Acetaminophen in Term Pregnancy." *American Journal of Perinatology* 34, no. 6 (May 2017): 541–43. https://doi.org/10.1055/s-0036-1593845.

Nordahl, Christine Wu, Daniel Braunschweig, Ana-Maria Iosif, Aaron Lee, Sally Rogers, Paul Ashwood, David G. Amaral, and Judy Van De Water. "Maternal Autoantibodies Are Associated with Abnormal Brain Enlargement in a Subgroup of Children with Autism Spectrum Disorder." *Brain, Behavior, and Immunity* 30 (May 2013): 61–65. https://doi.org/10.1016/j.bbi.2013.01.084.

Noriega, Daniela Briceno, and Huub F. J. Savelkoul. "Immune Dysregulation in Autism Spectrum Disorder." *European Journal of Pediatrics* 173, no. 1 (January 2014): 33–43. https://doi.org/10.1007/s00431-013-2183-4.

Norman, Luke J., Christina Carlisi, Steve Lukito, Heledd Hart, David Mataix-Cols, Joaquim Radua, and Katya Rubia. "Structural and Functional Brain Abnormalities in Attention-Deficit/Hyperactivity Disorder and Obsessive-Compulsive Disorder: A Comparative Meta-Analysis." *JAMA Psychiatry* 73, no. 8 (August 1, 2016): 815–25. https://doi.org/10.1001/jamapsychiatry.2016.0700.

Norris, Megan, Luc Lecavalier, and Michael C. Edwards. "The Structure of Autism Symptoms as Measured by the Autism Diagnostic Observation Schedule." *Journal of Autism and Developmental Disorders* 42, no. 6 (June 2012): 1075–86. https://doi.org/10.1007/s10803-011-1348-0.

Novotny, Sherie, Eric Hollander, Andrea Allen, Serge Mosovich, Bonnie Aronowitz, Charles Cartwright, Concetta DeCaria, and Rima Dolgoff-Kaspar. "Increased Growth Hormone Response to Sumatriptan Challenge in Adult Autistic Disorders." *Psychiatry Research* 94, no. 2 (May 2000): 173–77. https://doi.org/10.1016/S0165-1781(00)00134-7.

Novotny, Sherie, Eric Hollander, Ann Phillips, Andrea Allen, Stacey Wasserman, and Rupa Iyengar. “Increased Repetitive Behaviours and Prolactin Responsivity to Oral M-Chlorophenylpiperazine in Adults with Autism Spectrum Disorders.” *The International Journal of Neuropsychopharmacology* 7, no. 3 (September 2004): 249–54. https://doi.org/10.1017/S146114570400433X.

Oades, Robert D, Maria R Dauvermann, Benno G Schimmelmann, Markus J Schwarz, and Aye-Mu Myint. “Attention-Deficit Hyperactivity Disorder (ADHD) and Glial Integrity: S100B, Cytokines and Kynurenine Metabolism - Effects of Medication.” *Behavioral and Brain Functions : BBF* 6 (May 28, 2010): 29. https://doi.org/10.1186/1744-9081-6-29.

Oades, Robert D., Aye-Mu Myint, Maria R. Dauvermann, Benno G. Schimmelmann, and Markus J. Schwarz. “Attention-Deficit Hyperactivity Disorder (ADHD) and Glial Integrity: An Exploration of Associations of Cytokines and Kynurenine Metabolites with Symptoms and Attention.” *Behavioral and Brain Functions* 6, no. 1 (June 9, 2010): 32. https://doi.org/10.1186/1744-9081-6-32.

O’Brien, Gregory, and Joanne Pearson. “Autism and Learning Disability.” *Autism* 8, no. 2 (June 2004): 125–40. https://doi.org/10.1177/1362361304042718.

Oerbeck, Beate, Kristin Romvig Overgaard, Are Hugo Pripp, Ted Reichborn-Kjennerud, Heidi Aase, and Pal Zeiner. “Early Predictors of ADHD: Evidence from a Prospective Birth Cohort.” *Journal of Attention Disorders* 24, no. 12 (October 2020): 1685–92. https://doi.org/10.1177/1087054717696765.

Oken, Emily, Yi Ning, Sheryl L. Rifas-Shiman, Janet W. Rich-Edwards, Sjurdur F. Olsen, & Matthew W. Gillman. “Diet During Pregnancy and Risk of Preeclampsia or Gestational Hypertension.” *Annals of Epidemiology* 17, no. 9 (September 2007): 663–68. https://doi.org/10.1016/j.annepidem.2007.03.003.

Oláh, Attila, Arnold Markovics, Judit Szabó-Papp, Pálma Tímea Szabó, Colin Stott, Christos C. Zouboulis, and Tamás Bíró. “Differential Effectiveness of Selected Non-Psychotropic Phytocannabinoids on Human Sebocyte Functions Implicates Their Introduction in Dry/Seborrhoeic Skin and Acne Treatment.” *Experimental Dermatology* 25, no. 9 (September 2016): 701–7. https://doi.org/10.1111/exd.13042.

Olsen, Jørn, and Zeyan Liew. “Fetal Programming of Mental Health by Acetaminophen? Response to the SMFM Statement: Prenatal Acetaminophen Use and ADHD.” *Expert Opinion on Drug Safety* 16, no. 12 (December 2, 2017): 1395–98. https://doi.org/10.1080/14740338.2017.1384812.

Onaivi, E.S, R Benno, T Halpern, M Mehanovic, N Schanz, C Sanders, X Yan, et al. “Consequences of Cannabinoid and Monoaminergic System Disruption in a Mouse Model of Autism Spectrum Disorders.” *Current Neuropharmacology* 9, no. 1 (March 2011): 209–14. https://doi.org/10.2174/157015911795017047.

Onore, Charity, Milo Careaga, and Paul Ashwood. “The Role of Immune Dysfunction in the Pathophysiology of Autism.” *Brain, Behavior, and Immunity* 26, no. 3 (March 2012): 383–92. https://doi.org/10.1016/j.bbi.2011.08.007.

Onore, Charity E., Jared J. Schwartzer, Milo Careaga, Robert F. Berman, and Paul Ashwood. "Maternal Immune Activation Leads to Activated Inflammatory Macrophages in Offspring." *Brain, Behavior, and Immunity* 38 (May 2014): 220–26. https://doi.org/10.1016/j.bbi.2014.02.007.

Opris, Ioan, and Manuel F. Casanova. "Prefrontal Cortical Minicolumn: From Executive Control to Disrupted Cognitive Processing." *Brain* 137, no. 7 (July 2014): 1863–75. https://doi.org/10.1093/brain/awt359.

Orozco, Jennie Sotelo, Irva Hertz-Picciotto, Leonard Abbeduto, and Carolyn M. Slupsky. "Metabolomics Analysis of Children with Autism, Idiopathic-Developmental Delays, and Down Syndrome." *Translational Psychiatry* 9 (October 3, 2019): 243. https://doi.org/10.1038/s41398-019-0578-3.

O'Sullivan, Joseph, Alexandra E. Cairns, Elena Plesca, Rebecca S. Black, Charlotte Frise, Manu Vatish, and Ana Sofia Cerdeira. "Paracetamol Use in Pregnancy — Neglecting Context Promotes Misinterpretation." *Nature Reviews Endocrinology* 18, no. 6 (June 2022): 385–385. https://doi.org/10.1038/s41574-022-00656-9.

Ousley, Opal, and Tracy Cermak. "Autism Spectrum Disorder: Defining Dimensions and Subgroups." *Current Developmental Disorders Reports* 1, no. 1 (March 2014): 20–28. https://doi.org/10.1007/s40474-013-0003-1.

Owley, Thomas, Camille W. Brune, Jeff Salt, Laura Walton, Steve Guter, Nelson Ayuyao, Robert D. Gibbons, Bennett L. Leventhal, and Edwin H. Cook. "A Pharmacogenetic Study of Escitalopram in Autism Spectrum Disorders." *Autism Research*, 2009, n/a-n/a. https://doi.org/10.1002/aur.109.

Owley, Thomas, Laura Walton, Jeff Salt, Stephen J. Guter, Marrea Winnega, Bennett L. Leventhal, and Edwin H. Cook. "An Open-Label Trial of Escitalopram in Pervasive Developmental Disorders." *Journal of the American Academy of Child & Adolescent Psychiatry* 44, no. 4 (April 2005): 343–48. https://doi.org/10.1097/01.chi.0000153229.80215.a0.

Pallanti, Stefano, Lorenza Bencini, Andrea Cantisani, and Eric Hollander. "Psychotropic Treatment of Autism." In *Key Issues in Mental Health*, edited by M. Leboyer and P. Chaste, 180:151–65. S. Karger AG, 2015. https://doi.org/10.1159/000363614.

Panagiotou, Maria, Kostas Papagiannopoulos, Jos H. T. Rohling, Johanna H. Meijer, and Tom Deboer. "How Old Is Your Brain? Slow-Wave Activity in Non-Rapid-Eye-Movement Sleep as a Marker of Brain Rejuvenation After Long-Term Exercise in Mice." *Frontiers in Aging Neuroscience* 10 (August 7, 2018): 233. https://doi.org/10.3389/fnagi.2018.00233.

Panagiotou, Orestis A., Despina G. Contopoulos-Ioannidis, Panagiotis N. Papanikolaou, Evangelia E. Ntzani, and John P. A. Ioannidis. "Different Black Box Warning Labeling for Same-Class Drugs." *Journal of General Internal Medicine* 26, no. 6 (June 2011): 603–10. https://doi.org/10.1007/s11606-011-1633-9.

Pandina, Gahan J., Cynthia A. Bossie, Eriene Youssef, Young Zhu, and Fiona Dunbar. "Risperidone Improves Behavioral Symptoms in Children with Autism in a Randomized, Double-Blind, Placebo-Controlled Trial." *Journal of Autism and Developmental Disorders* 37, no. 2 (February 22, 2007): 367–73. https://doi.org/10.1007/s10803-006-0234-7.

Papageorgiou, Vaya, Efrosini Kalyva, Vaios Dafoulis, and Panos Vostanis. "Differences in Parents' and Teachers' Ratings of ADHD Symptoms and Other Mental Health Problems." *European Journal of Psychiatry - EUR J PSYCHIAT* 22 (October 1, 2008). https://doi.org/10.4321/S0213-61632008000400003.

Pardo, Carlos A, Ashura Buckley, Audrey Thurm, Li-Ching Lee, Arun Azhagiri, David M Neville, and Susan E Swedo. "A Pilot Open-Label Trial of Minocycline in Patients with Autism and Regressive Features." *Journal of Neurodevelopmental Disorders* 5, no. 1 (December 2013): 9. https://doi.org/10.1186/1866-1955-5-9.

Pardo, Carlos A., Diana L. Vargas, and Andrew W. Zimmerman. "Immunity, Neuroglia and Neuroinflammation in Autism." *International Review of Psychiatry* 17, no. 6 (January 2005): 485–95. https://doi.org/10.1080/02646830500381930.

Park, Hye Ran, Jae Meen Lee, Hyo Eun Moon, Dong Soo Lee, Bung-Nyun Kim, Jinhyun Kim, Dong Gyu Kim, and Sun Ha Paek. "A Short Review on the Current Understanding of Autism Spectrum Disorders." *Experimental Neurobiology* 25, no. 1 (February 29, 2016): 1–13. https://doi.org/10.5607/en.2016.25.1.1.

Parker, Karen J., Ozge Oztan, Robin A. Libove, Noreen Mohsin, Debra S. Karhson, Raena D. Sumiyoshi, Jacqueline E. Summers, et al. "A Randomized Placebo-Controlled Pilot Trial Shows That Intranasal Vasopressin Improves Social Deficits in Children with Autism." *Science Translational Medicine* 11, no. 491 (May 8, 2019): eaau7356. https://doi.org/10.1126/scitranslmed.aau7356.

Parker, Samantha E., Brent R. Collett, and Martha M. Werler. "Maternal Acetaminophen Use during Pregnancy and Childhood Behavioural Problems: Discrepancies between Mother- and Teacher-reported Outcomes." *Paediatric and Perinatal Epidemiology* 34, no. 3 (May 2020): 299–308. https://doi.org/10.1111/ppe.12601.

Parker, William, Chi Dang Hornik, Staci Bilbo, Zoie E. Holzknecht, Lauren Gentry, Rasika Rao, Shu S. Lin, Martha R. Herbert, and Cynthia D. Nevison. "The Role of Oxidative Stress, Inflammation and Acetaminophen Exposure from Birth to Early Childhood in the Induction of Autism." *The Journal of International Medical Research* 45, no. 2 (April 2017): 407–38. https://doi.org/10.1177/0300060517693423.

Patterson, Paul H. "Maternal Infection and Autism." *Brain, Behavior, and Immunity* 26, no. 3 (March 2012): 393. https://doi.org/10.1016/j.bbi.2011.09.008.

Pedersen, Kahsi A., Susan L. Santangelo, Robin L. Gabriels, Giulia Righi, Michael Erard, and Matthew Siegel. "Behavioral Outcomes of Specialized Psychiatric Hospitalization in the Autism Inpatient Collection (AIC): A Multisite Comparison." *Journal of Autism and Developmental Disorders* 48, no. 11 (November 2018): 3658–67. https://doi.org/10.1007/s10803-017-3366-z.

Pelton, Mirabel K., Hayley Crawford, Ashley E. Robertson, Jacqui Rodgers, Simon Baron-Cohen, and Sarah Cassidy. "Understanding Suicide Risk in Autistic Adults: Comparing the Interpersonal Theory of Suicide in Autistic and Non-Autistic Samples." *Journal of Autism and Developmental Disorders* 50, no. 10 (October 2020): 3620–37. https://doi.org/10.1007/s10803-020-04393-8.

Peñagarikano, Olga, and Daniel H. Geschwind. "What Does CNTNAP2 Reveal about Autism Spectrum Disorder?" *Trends in Molecular Medicine* 18, no. 3 (March 2012): 156–63. https://doi.org/10.1016/j.molmed.2012.01.003.

Pernia, Sonia, and George DeMaagd. "The New Pregnancy and Lactation Labeling Rule." *Pharmacy and Therapeutics* 41, no. 11 (November 2016): 713–15.

Pezzimenti, Florencia, Gloria T. Han, Roma A. Vasa, and Katherine Gotham. "Depression in Youth with Autism Spectrum Disorder." *Child and Adolescent Psychiatric Clinics of North America* 28, no. 3 (July 2019): 397–409. https://doi.org/10.1016/j.chc.2019.02.009.

Picci, Giorgia, and K. Suzanne Scherf. "A Two-Hit Model of Autism: Adolescence as the Second Hit." *Clinical Psychological Science : A Journal of the Association for Psychological Science* 3, no. 3 (May 2015): 349–71. https://doi.org/10.1177/2167702614540646.

Pineda, Eduardo, Don Shin, Su Jeong You, Stéphane Auvin, Raman Sankar, and Andréy Mazarati. "Maternal Immune Activation Promotes Hippocampal Kindling Epileptogenesis in Mice: Pineda et al: MIA and Epilepsy." *Annals of Neurology* 74, no. 1 (July 2013): 11–19. https://doi.org/10.1002/ana.23898.

Pinto, Dalila, Elsa Delaby, Daniele Merico, Mafalda Barbosa, Alison Merikangas, Lambertus Klei, Bhooma Thiruvahindrapuram, et al. "Convergence of Genes and Cellular Pathways Dysregulated in Autism Spectrum Disorders." *The American Journal of Human Genetics* 94, no. 5 (May 2014): 677–94. https://doi.org/10.1016/j.ajhg.2014.03.018.

Polanczyk, Guilherme, Maurício Silva de Lima, Bernardo Lessa Horta, Joseph Biederman, and Luis Augusto Rohde. "The Worldwide Prevalence of ADHD: A Systematic Review and Metaregression Analysis." *Am J Psychiatry*, 2007.

Polanczyk, Guilherme V, Erik G Willcutt, Giovanni A Salum, Christian Kieling, and Luis A Rohde. "ADHD Prevalence Estimates across Three Decades: An Updated Systematic Review and Meta-Regression Analysis." *International Journal of Epidemiology* 43, no. 2 (April 2014): 434–42. https://doi.org/10.1093/ije/dyt261.

Poljac, Edita, Vincent Hoofs, Myrthe M. Princen, and Ervin Poljac. "Understanding Behavioural Rigidity in Autism Spectrum Conditions: The Role of Intentional Control." *Journal of Autism and Developmental Disorders* 47, no. 3 (March 2017): 714–27. https://doi.org/10.1007/s10803-016-3010-3.

Posadas, Inmaculada, Pablo Santos, Almudena Blanco, Maríangeles Muñoz-Fernández, and Valentín Ceña. "Acetaminophen Induces Apoptosis in Rat Cortical Neurons." *PloS One* 5, no. 12 (December 10, 2010): e15360. https://doi.org/10.1371/journal.pone.0015360.

Posner, Jonathan, Guilherme V Polanczyk, and Edmund Sonuga-Barke. “Attention-Deficit Hyperactivity Disorder.” *Lancet (London, England)* 395, no. 10222 (February 8, 2020): 450–62. https://doi.org/10.1016/S0140-6736(19)33004-1.

“Prevalence of ADHD and Its Comorbidities in a Population-Based Sample.” Accessed May 18, 2023. https://doi.org/10.1177/1087054719886372.

“Psychiatric Consultation in Severe and Profound Mental Retardation.” *American Journal of Psychiatry* 151, no. 12 (December 1994): 1802–8. https://doi.org/10.1176/ajp.151.12.1802.

Pugsley, Kealan, Stephen W. Scherer, Mark A. Bellgrove, and Ziarih Hawi. “Environmental Exposures Associated with Elevated Risk for Autism Spectrum Disorder May Augment the Burden of Deleterious de Novo Mutations among Probands.” *Molecular Psychiatry* 27, no. 1 (January 2022): 710–30. https://doi.org/10.1038/s41380-021-01142-w.

Purper-Ouakil, Diane, Nicolas Ramoz, Aude-Marie Lepagnol-Bestel, Philip Gorwood, and Michel Simonneau. “Neurobiology of Attention Deficit/Hyperactivity Disorder.” *Pediatric Research* 69, no. 8 (May 2011): 69–76. https://doi.org/10.1203/PDR.0b013e318212b40f.

Rahil R. Jummani, M. D., Emily Hirsch, and M. D. Glenn S. Hirsch. “Are We Overdiagnosing and Overtreating ADHD?,” Vol 34 No 5, 34 (May 31, 2017). https://www.psychiatrictimes.com/view/are-we-overdiagnosing-and-overtreating-adhd.

Rai-Bhogal, Ravneet, Eizaaz Ahmad, Hongyan Li, and Dorota A. Crawford. “Microarray Analysis of Gene Expression in the Cyclooxygenase Knockout Mice - a Connection to Autism Spectrum Disorder.” *The European Journal of Neuroscience* 47, no. 6 (March 2018): 750–66. https://doi.org/10.1111/ejn.13781.

Raja, Michele. “AUTISM Spectrum Disorders and Suicidality.” *Clinical Practice § Epidemiology in Mental Health* 7, no. 1 (March 30, 2011): 97–105. https://doi.org/10.2174/1745017901107010097.

Ramoz, Nicolas, Guiqing Cai, Jennifer G. Reichert, Thomas E. Corwin, Lauren A. Kryzak, Christopher J. Smith, Jeremy M. Silverman, Eric Hollander, and Joseph D. Buxbaum. “Family-Based Association Study of TPH1 and TPH2 Polymorphisms in Autism.” *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* 141B, no. 8 (December 5, 2006): 861–67. https://doi.org/10.1002/ajmg.b.30356.

Ramoz, Nicolas, Jennifer G. Reichert, Thomas E. Corwin, Christopher J. Smith, Jeremy M. Silverman, Eric Hollander, and Joseph D. Buxbaum. “Lack of Evidence for Association of the Serotonin Transporter Gene SLC6A4 with Autism.” *Biological Psychiatry* 60, no. 2 (July 2006): 186–91. https://doi.org/10.1016/j.biopsych.2006.01.009.

Ramtekkar, Ujjwal P., Angela M. Reiersen, Alexandre A. Todorov, and Richard D. Todd. “Sex and Age Differences in Attention-Deficit/Hyperactivity Disorder Symptoms and Diagnoses: Implications for DSM-V and ICD-11.” *Journal of the American Academy of Child and Adolescent Psychiatry* 49, no. 3 (March 2010): 217-28.e1-3.

Rebordosa, Cristina, Carolyn M. Zelop, Manolis Kogevinas, Henrik T. Sørensen, and Jørn Olsen. "Use of Acetaminophen during Pregnancy and Risk of Preeclampsia, Hypertensive and Vascular Disorders: A Birth Cohort Study." *The Journal of Maternal-Fetal & Neonatal Medicine: The Official Journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians* 23, no. 5 (May 2010): 371–78. https://doi.org/10.3109/14767050903334877.

Reichard, Julia, and Geraldine Zimmer-Bensch. "The Epigenome in Neurodevelopmental Disorders." *Frontiers in Neuroscience* 15 (November 3, 2021): 776809. https://doi.org/10.3389/fnins.2021.776809.

Reiersen, Angela M., John N. Constantino, Heather E. Volk, and Richard D. Todd. "Autistic Traits in a Population-Based ADHD Twin Sample." *Journal of Child Psychology and Psychiatry* 48, no. 5 (2007): 464–72. https://doi.org/10.1111/j.1469-7610.2006.01720.x.

Ressel, Madelaine, Brittni Thompson, Marie-Hélène Poulin, Claude L Normand, Marissa H Fisher, Germain Couture, and Grace Iarocci. "Systematic Review of Risk and Protective Factors Associated with Substance Use and Abuse in Individuals with Autism Spectrum Disorders." *Autism* 24, no. 4 (May 2020): 899–918. https://doi.org/10.1177/1362361320910963.

Restrepo, Anita, Tohar Scheininger, Jon Clucas, Lindsay Alexander, Giovanni A. Salum, Kathy Georgiades, Diana Paksarian, Kathleen R. Merikangas, and Michael P. Milham. "Problematic Internet Use in Children and Adolescents: Associations with Psychiatric Disorders and Impairment." *BMC Psychiatry* 20, no. 1 (December 2020): 252. https://doi.org/10.1186/s12888-020-02640-x.

Ricci, Christina, Carmela Melina Albanese, Lesley A. Pablo, Jiaying Li, Maryam Fatima, Kathryn Barrett, Brooke Levis, and Hilary K. Brown. "In Utero Acetaminophen Exposure and Child Neurodevelopmental Outcomes: Systematic Review and Meta-Analysis." *Paediatric and Perinatal Epidemiology* n/a, no. n/a (2023). https://doi.org/10.1111/ppe.12963.

Rice, D, and S Barone. "Critical Periods of Vulnerability for the Developing Nervous System: Evidence from Humans and Animal Models." *Environmental Health Perspectives* 108, no. Suppl 3 (June 2000): 511–33.

Richards, Gareth, Rebecca Kenny, Sarah Griffiths, Carrie Allison, David Mosse, Rosemary Holt, Rory C. O'Connor, Sarah Cassidy, and Simon Baron-Cohen. "Autistic Traits in Adults Who Have Attempted Suicide." *Molecular Autism* 10, no. 1 (December 2019): 26. https://doi.org/10.1186/s13229-019-0274-4.

Richler, Jennifer, Marisela Huerta, Somer L. Bishop, and Catherine Lord. "Developmental Trajectories of Restricted and Repetitive Behaviors and Interests in Children with Autism Spectrum Disorders." *Development and Psychopathology* 22, no. 1 (2010): 55–69. https://doi.org/10.1017/S0954579409990265.

Rifas-Shiman, Sheryl L., Andres Cardenas, Marie-France Hivert, Henning Tiemeier, Andrea D. Bertoldi, and Emily Oken. “Associations of Prenatal or Infant Exposure to Acetaminophen or Ibuprofen with Mid-childhood Executive Function and Behaviour.” *Paediatric and Perinatal Epidemiology* 34, no. 3 (May 2020): 287–98. https://doi.org/10.1111/ppe.12596.

Righi, Giula, Giulia Righi, Jill Benevides, Carla Mazefsky, Matthew Siegel, Stephen J. Sheinkopf, and Eric M. Morrow. “Predictors of Inpatient Psychiatric Hospitalization for Children and Adolescents with Autism Spectrum Disorder.” *Journal of Autism and Developmental Disorders* 48, no. 11 (November 2018): 3647–57. https://doi.org/10.1007/s10803-017-3154-9.

“Risperidone in Children with Autism and Serious Behavioral Problems.” *The New England Journal of Medicine*, 2002.

Ritvo, E R. “Autism Treatments Proposed by Clinical Studies and Human Genetics Are Complementary.” *Translational Psychiatry* 4, no. 7 (July 29, 2014): e415–e415. https://doi.org/10.1038/tp.2014.32.

Roane, Henry S., Wayne W. Fisher, and James E. Carr. “Applied Behavior Analysis as Treatment for Autism Spectrum Disorder.” *The Journal of Pediatrics* 175 (August 1, 2016): 27–32. https://doi.org/10.1016/j.jpeds.2016.04.023.

Robb, Adelaide S. “Managing Irritability and Aggression in Autism Spectrum Disorders in Children and Adolescents.” *Developmental Disabilities Research Reviews* 16, no. 3 (2010): 258–64. https://doi.org/10.1002/ddrr.118.

Robbins, Jennifer R., and Anna I. Bakardjiev. “Pathogens and the Placental Fortress.” *Current Opinion in Microbiology* 15, no. 1 (February 2012): 36–43. https://doi.org/10.1016/j.mib.2011.11.006.

Roberts, Emmert, Vanessa Delgado Nunes, Sara Buckner, Susan Latchem, Margaret Constanti, Paul Miller, Michael Doherty, et al. “Paracetamol: Not as Safe as We Thought? A Systematic Literature Review of Observational Studies.” *Annals of the Rheumatic Diseases* 75, no. 3 (March 2016): 552–59. https://doi.org/10.1136/annrheumdis-2014-206914.

Rock, Erin M, Martin A Sticht, Marnie Duncan, Colin Stott, and Linda A Parker. “Evaluation of the Potential of the Phytocannabinoids, Cannabidivarin (CBDV) and Δ^{9} -Tetrahydrocannabivarin (THCV), to Produce CB_{1} Receptor Inverse Agonism Symptoms of Nausea in Rats: THCV and CBDV and Nausea.” *British Journal of Pharmacology* 170, no. 3 (October 2013): 671–78. https://doi.org/10.1111/bph.12322.

Rodier, P M. “Developing Brain as a Target of Toxicity.” *Environmental Health Perspectives* 103, no. Suppl 6 (September 1995): 73–76.

Rodier, P. M., J. L. Ingram, B. Tisdale, S. Nelson, and J. Romano. “Embryological Origin for Autism: Developmental Anomalies of the Cranial Nerve Motor Nuclei.” *The Journal of Comparative Neurology* 370, no. 2 (June 24, 1996): 247–61. https://doi.org/10.1002/(SICI)1096-9861(19960624)370:2<247::AID-CNE8>3.0.CO;2-2.

Rodrigues, Rebecca, Meng-Chuan Lai, Adam Beswick, Daniel A. Gorman, Evdokia Anagnostou, Peter Szatmari, Kelly K. Anderson, and Stephanie H. Ameis. “Practitioner Review: Pharmacological Treatment of Attention-deficit/Hyperactivity Disorder Symptoms in Children and Youth with Autism Spectrum Disorder: A Systematic Review and Meta-analysis.” *Journal of Child Psychology and Psychiatry* 62, no. 6 (June 2021): 680–700. https://doi.org/10.1111/jcpp.13305.

Rodriguez, Juan I., and Janet K. Kern. “Evidence of Microglial Activation in Autism and Its Possible Role in Brain Underconnectivity.” *Neuron Glia Biology* 7, no. 2–4 (May 2011): 205–13. https://doi.org/10.1017/S1740925X12000142.

Rojahn, Johannes, Scott T Matlock, and Marc J Tassé. “The Stereotyped Behavior Scale: Psychometric Properties and Norms.” *Research in Developmental Disabilities* 21, no. 6 (November 2000): 437–54. https://doi.org/10.1016/S0891-4222(00)00057-3.

Rommel, Anna-Sophie, Ginger L. Milne, Emily S. Barrett, Nicole R. Bush, Ruby Nguyen, Sheela Sathyanarayana, Shanna H. Swan, and Kelly K. Ferguson. “Associations between Urinary Biomarkers of Oxidative Stress in the Third Trimester of Pregnancy and Behavioral Outcomes in the Child at 4 Years of Age.” *Brain, Behavior, and Immunity* 90 (November 2020): 272–78. https://doi.org/10.1016/j.bbi.2020.08.029.

Rommelse, Nanda, Ilse Langerak, Jolanda Van Der Meer, Yvette De Bruijn, Wouter Staal, Anoek Oerlemans, and Jan Buitelaar. “Intelligence May Moderate the Cognitive Profile of Patients with ASD.” Edited by Sam Gilbert. *PLOS ONE* 10, no. 10 (October 7, 2015): e0138698. https://doi.org/10.1371/journal.pone.0138698.

Ronemus, Michael, Ivan Iossifov, Dan Levy, and Michael Wigler. “The Role of de Novo Mutations in the Genetics of Autism Spectrum Disorders.” *Nature Reviews Genetics* 15, no. 2 (February 2014): 133–41. https://doi.org/10.1038/nrg3585.

Rook, Graham A.W., Christopher A. Lowry, and Charles L. Raison. “Hygiene and Other Early Childhood Influences on the Subsequent Function of the Immune System.” *Brain Research* 1617 (August 2015): 47–62. https://doi.org/10.1016/j.brainres.2014.04.004.

Rose, Destanie, and Paul Ashwood. “Potential Cytokine Biomarkers in Autism Spectrum Disorders.” *Biomarkers in Medicine* 8, no. 9 (2014): 1171–81. https://doi.org/10.2217/bmm.14.39.

Rose, S, S Melnyk, O Pavliv, S Bai, T G Nick, R E Frye, and S J James. “Evidence of Oxidative Damage and Inflammation Associated with Low Glutathione Redox Status in the Autism Brain.” *Translational Psychiatry* 2, no. 7 (July 2012): e134. https://doi.org/10.1038/tp.2012.61.

Rucklidge, Julia J. “Gender Differences in Attention-Deficit/Hyperactivity Disorder.” *Psychiatric Clinics of North America* 33, no. 2 (June 2010): 357–73. https://doi.org/10.1016/j.psc.2010.01.006.

Ruisch, I. Hyun, Jan K. Buitelaar, Jeffrey C. Glennon, Pieter J. Hoekstra, and Andrea Dietrich. “Pregnancy Risk Factors in Relation to Oppositional-Defiant and Conduct Disorder Symptoms in the Avon Longitudinal Study of Parents and Children.” *Journal of Psychiatric Research* 101 (June 2018): 63–71.

https://doi.org/10.1016/j.jpsychires.2018.02.020.

Russell, Ginny, Lauren R. Rodgers, and Tamsin Ford. "The Strengths and Difficulties Questionnaire as a Predictor of Parent-Reported Diagnosis of Autism Spectrum Disorder and Attention Deficit Hyperactivity Disorder." Edited by Atsushi Senju. *PLoS ONE* 8, no. 12 (December 3, 2013): e80247. https://doi.org/10.1371/journal.pone.0080247.

Ryan, Duncan, Alison J. Drysdale, Carlos Lafourcade, Roger G. Pertwee, and Bettina Platt. "Cannabidiol Targets Mitochondria to Regulate Intracellular Ca2+ Levels." *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience* 29, no. 7 (February 18, 2009): 2053–63. https://doi.org/10.1523/JNEUROSCI.4212-08.2009.

Sabapathy, Thusa, Douglas L. Vanderbilt, Irina Zamora, and Marilyn Augustyn. "Aggression in Autism Spectrum Disorder: Supporting the Entire Family." *Journal of Developmental & Behavioral Pediatrics* 37, no. 8 (October 2016): 685–86. https://doi.org/10.1097/DBP.0000000000000347.

Sachdeva, Punam, B. G. Patel, and B. K. Patel. "Drug Use in Pregnancy; a Point to Ponder!" *Indian Journal of Pharmaceutical Sciences* 71, no. 1 (2009): 1–7. https://doi.org/10.4103/0250-474X.51941.

Sádecký, E., R. Brezina, J. Kazár, and J. Urvölgyi. "Immunization against Q-Fever of Naturally Infected Dairy Cows." *Acta Virologica* 19, no. 6 (November 1975): 486–88.

Sakurai, Takeshi, Nicolas Ramoz, Jennifer G. Reichert, Thomas E. Corwin, Lauren Kryzak, Christopher J. Smith, Jeremy M. Silverman, Eric Hollander, and Joseph D. Buxbaum. "Association Analysis of the NrCAM Gene in Autism and in Subsets of Families with Severe Obsessive–Compulsive or Self-Stimulatory Behaviors." *Psychiatric Genetics* 16, no. 6 (December 2006): 251–57. https://doi.org/10.1097/01.ypg.0000242196.81891.c9.

Salcedo-Marin, M. D., J. M. Moreno-Granados, M. Ruiz-Veguilla, and M. Ferrin. "Evaluation of Planning Dysfunction in Attention Deficit Hyperactivity Disorder and Autistic Spectrum Disorders Using the Zoo Map Task." *Child Psychiatry & Human Development* 44, no. 1 (February 2013): 166–85. https://doi.org/10.1007/s10578-012-0317-y.

Sampino, S., G. R. Juszczak, F. Zacchini, A. H. Swiergiel, J. A. Modlinski, P. Loi, and G. E. Ptak. "Grand-Paternal Age and the Development of Autism-like Symptoms in Mice Progeny." *Translational Psychiatry* 4, no. 4 (April 2014): e386–e386. https://doi.org/10.1038/tp.2014.27.

Sandin, S., D. Schendel, P. Magnusson, C. Hultman, P. Surén, E. Susser, T. Grønborg, et al. "Autism Risk Associated with Parental Age and with Increasing Difference in Age between the Parents." *Molecular Psychiatry* 21, no. 5 (May 2016): 693–700. https://doi.org/10.1038/mp.2015.70.

Sandin, Sven, Paul Lichtenstein, Ralf Kuja-Halkola, Henrik Larsson, Christina M. Hultman, and Abraham Reichenberg. "The Familial Risk of Autism." *JAMA* 311, no. 17 (May 7, 2014): 1770–77. https://doi.org/10.1001/jama.2014.4144.

Sangha, Susan, Quentin Greba, Paul D. Robinson, Stephanie A. Ballendine, and John G. Howland. "Heightened Fear in Response to a Safety Cue and Extinguished Fear Cue in a Rat Model of Maternal Immune Activation." *Frontiers in Behavioral Neuroscience* 8 (May 7, 2014). https://doi.org/10.3389/fnbeh.2014.00168.

Santosh, Paramala J, and Angela Mijovic. "Does Pervasive Developmental Disorder Protect Children and Adolescents against Drug and Alcohol Use?" *European Child & Adolescent Psychiatry* 15, no. 4 (June 2006): 183–88. https://doi.org/10.1007/s00787-005-0517-0.

Sayal, Kapil, Vibhore Prasad, David Daley, Tamsin Ford, and David Coghill. "ADHD in Children and Young People: Prevalence, Care Pathways, and Service Provision." *The Lancet Psychiatry* 5, no. 2 (February 2018): 175–86. https://doi.org/10.1016/S2215-0366(17)30167-0.

Scahill, Lawrence, Michael G Aman, Luc Lecavalier, Alycia K Halladay, Somer L Bishop, James W Bodfish, Sabrina Grondhuis, et al. "Measuring Repetitive Behaviors as a Treatment Endpoint in Youth with Autism Spectrum Disorder." *Autism* 19, no. 1 (January 2015): 38–52. https://doi.org/10.1177/1362361313510069.

Scahill, Lawrence, Anastasia Dimitropoulos, Christopher J. McDougle, Michael G. Aman, Irene D. Feurer, James T. McCracken, Elaine Tierney, et al. "Children's Yale–Brown Obsessive Compulsive Scale in Autism Spectrum Disorder: Component Structure and Correlates of Symptom Checklist." *Journal of the American Academy of Child & Adolescent Psychiatry* 53, no. 1 (January 2014): 97-107.e1. https://doi.org/10.1016/j.jaac.2013.09.018.

Scahill, Lawrence, Christopher J. Mcdougle, Susan K. Williams, Anastasia Dimitropoulos, Michael G. Aman, James T. Mccracken, Elaine Tierney, et al. "Children's Yale-Brown Obsessive Compulsive Scale Modified for Pervasive Developmental Disorders." *Journal of the American Academy of Child & Adolescent Psychiatry* 45, no. 9 (September 2006): 1114–23. https://doi.org/10.1097/01.chi.0000220854.79144.e7.

Scahill, Lawrence, Mark A. Riddle, Maureen McSWIGGIN-HARDIN, Sharon I. Ort, Robert A. King, Wayne K. Goodman, Domenic Cicchetti, and James F. Leckman. "Children's Yale-Brown Obsessive Compulsive Scale: Reliability and Validity." *Journal of the American Academy of Child & Adolescent Psychiatry* 36, no. 6 (June 1997): 844–52. https://doi.org/10.1097/00004583-199706000-00023.

Scahill, Lawrence, Denis G Sukhodolsky, Emily Anderberg, Anastasia Dimitropoulos, James Dziura, Michael G Aman, James McCracken, et al. "Sensitivity of the Modified Children's Yale–Brown Obsessive Compulsive Scale to Detect Change: Results from Two Multi-Site Trials." *Autism* 20, no. 2 (February 2016): 145–52. https://doi.org/10.1177/1362361315574889.

Schans, Jurjen van der, Rukiye Çiçek, Tjalling W. de Vries, Eelko Hak, and Pieter J. Hoekstra. "Association of Atopic Diseases and Attention-Deficit/Hyperactivity Disorder: A Systematic Review and Meta-Analyses." *Neuroscience & Biobehavioral Reviews* 74 (March 1, 2017): 139–48. https://doi.org/10.1016/j.neubiorev.2017.01.011.

Scharfstein, Lindsay A., Deborah C. Beidel, Valerie K. Sims, and Laura Rendon Finnell. "Social Skills Deficits and Vocal Characteristics of Children with Social Phobia or Asperger's Disorder: A Comparative Study." *Journal of Abnormal Child Psychology* 39, no. 6 (August 2011): 865–75. https://doi.org/10.1007/s10802-011-9498-2.

Scheerer, Nichole E., Anahid Pourtousi, Connie Yang, Zining Ding, Bobby Stojanoski, Evdokia Anagnostou, Robert Nicolson, et al. "Transdiagnostic Patterns of Sensory Processing in Autism and ADHD." *Journal of Autism and Developmental Disorders*, October 28, 2022. https://doi.org/10.1007/s10803-022-05798-3.

Schendel, Diana, and Tanya Karapurkar Bhasin. "Birth Weight and Gestational Age Characteristics of Children with Autism, Including a Comparison with Other Developmental Disabilities." *Pediatrics* 121, no. 6 (June 2008): 1155–64. https://doi.org/10.1542/peds.2007-1049.

Schendel, Diana E., Morten Overgaard, Jakob Christensen, Lene Hjort, Meta Jørgensen, Mogens Vestergaard, and Erik T. Parner. "Association of Psychiatric and Neurologic Comorbidity With Mortality Among Persons With Autism Spectrum Disorder in a Danish Population." *JAMA Pediatrics* 170, no. 3 (March 1, 2016): 243. https://doi.org/10.1001/jamapediatrics.2015.3935.

Schmitt, J., A. Buske-Kirschbaum, and V. Roessner. "Is Atopic Disease a Risk Factor for Attention-Deficit/Hyperactivity Disorder? A Systematic Review." *Allergy* 65, no. 12 (December 2010): 1506–24. https://doi.org/10.1111/j.1398-9995.2010.02449.x.

Schnider, Patrick, Caterina Bissantz, Andreas Bruns, Cosimo Dolente, Erwin Goetschi, Roland Jakob-Roetne, Basil Künnecke, et al. "Discovery of Balovaptan, a Vasopressin 1a Receptor Antagonist for the Treatment of Autism Spectrum Disorder." *Journal of Medicinal Chemistry* 63, no. 4 (February 27, 2020): 1511–25. https://doi.org/10.1021/acs.jmedchem.9b01478.

Schothorst, P. F., and H. van Engeland. "Long-Term Behavioral Sequelae of Prematurity." *Journal of the American Academy of Child and Adolescent Psychiatry* 35, no. 2 (February 1996): 175–83. https://doi.org/10.1097/00004583-199602000-00011.

Schöttle, Daniel, Peer Briken, Oliver Tüscher, and Daniel Turner. "Sexuality in Autism: Hypersexual and Paraphilic Behavior in Women and Men with High-Functioning Autism Spectrum Disorder." *Dialogues in Clinical Neuroscience* 19, no. 4 (December 31, 2017): 381–93. https://doi.org/10.31887/DCNS.2017.19.4/dschoettle.

Schultz, Stephen, Mauris DeSilva, Ting Ting Gu, Mei Qiang, and Kyumin Whang. "Effects of the Analgesic Acetaminophen (Paracetamol) and Its Para-Aminophenol Metabolite on Viability of Mouse-Cultured Cortical Neurons." *Basic & Clinical Pharmacology & Toxicology* 110, no. 2 (February 2012): 141–44. https://doi.org/10.1111/j.1742-7843.2011.00767.x.

Schultz, Stephen, Georgianna G. Gould, Nicola Antonucci, Anna Lisa Brigida, and Dario Siniscalco. "Endocannabinoid System Dysregulation from Acetaminophen Use May Lead to Autism Spectrum Disorder: Could Cannabinoid Treatment Be Efficacious?" *Molecules* 26, no. 7 (March 25, 2021): 1845. https://doi.org/10.3390/molecules26071845.

Schultz, Stephen, Dario Siniscalco, Stephen Schultz, and Dario Siniscalco. "Endocannabinoid System Involvement in Autism Spectrum Disorder: An Overview with Potential Therapeutic Applications." *AIMS Molecular Science* 6, no. 1 (2019): 27–37. https://doi.org/10.3934/molsci.2019.1.27.

Schultz, Stephen T. "Does Thimerosal or Other Mercury Exposure Increase the Risk for Autism?," 2010, 9.

Schultz, Stephen T, and Georgianna G Gould. "Acetaminophen Use for Fever in Children Associated with Autism Spectrum Disorder." *Autism-Open Access* 6, no. 2 (April 2016): 170. https://doi.org/10.4172/2165-7890.1000170.

Schwartzer, J J, M Careaga, C E Onore, J A Rushakoff, R F Berman, and P Ashwood. "Maternal Immune Activation and Strain Specific Interactions in the Development of Autism-like Behaviors in Mice." *Translational Psychiatry* 3, no. 3 (March 12, 2013): e240–e240. https://doi.org/10.1038/tp.2013.16.

Scott, Fiona J., Simon Baron-Cohen, Patrick Bolton, and Carol Brayne. "The CAST (Childhood Asperger Syndrome Test): Preliminary Development of a UK Screen for Mainstream Primary-School-Age Children." *Autism* 6, no. 1 (March 1, 2002): 9–31. https://doi.org/10.1177/1362361302006001003.

Scumpia, Philip O., Kindra Kelly-Scumpia, and Bruce R. Stevens. "Alpha-Lipoic Acid Effects on Brain Glial Functions Accompanying Double-Stranded RNA Antiviral and Inflammatory Signaling." *Neurochemistry International* 64 (January 2014): 55–63. https://doi.org/10.1016/j.neuint.2013.11.006.

Selden, Paul A., and Jakob Krieger. "Editorial." *Arthropod Structure & Development* 64 (September 2021): 101099. https://doi.org/10.1016/j.asd.2021.101099.

Sharma, Alok, and Justin Couture. "A Review of the Pathophysiology, Etiology, and Treatment of Attention-Deficit Hyperactivity Disorder (ADHD)." *The Annals of Pharmacotherapy* 48, no. 2 (February 2014): 209–25. https://doi.org/10.1177/1060028013510699.

Shelton, Janie F., Estella M. Geraghty, Daniel J. Tancredi, Lora D. Delwiche, Rebecca J. Schmidt, Beate Ritz, Robin L. Hansen, and Irva Hertz-Picciotto. "Neurodevelopmental Disorders and Prenatal Residential Proximity to Agricultural Pesticides: The CHARGE Study." *Environmental Health Perspectives* 122, no. 10 (October 2014): 1103–9. https://doi.org/10.1289/ehp.1307044.

Shen, Chun, Qiang Luo, Tianye Jia, Qi Zhao, Sylvane Desrivières, Erin Burke Quinlan, Tobias Banaschewski, et al. "Neural Correlates of the Dual-Pathway Model for ADHD in Adolescents." *American Journal of Psychiatry* 177, no. 9 (September 1, 2020): 844–54. https://doi.org/10.1176/appi.ajp.2020.19020183.

Shephard, Elizabeth, Bosiljka Milosavljevic, Greg Pasco, Emily J. H. Jones, Teodora Gliga, Francesca Happé, Mark H. Johnson, Tony Charman, and The BASIS Team. "Mid-Childhood Outcomes of Infant Siblings at Familial High-Risk of Autism Spectrum Disorder: Mid-Childhood Outcomes of HR Infant Siblings." *Autism Research* 10, no. 3 (March 2017): 546–57. https://doi.org/10.1002/aur.1733.

Shi, Limin, Stephen E.P. Smith, Natalia Malkova, Doris Tse, Yixuan Su, and Paul H. Patterson. "Activation of the Maternal Immune System Alters Cerebellar Development in the Offspring." *Brain, Behavior, and Immunity* 23, no. 1 (January 2009): 116–23. https://doi.org/10.1016/j.bbi.2008.07.012.

Shields, Morgan C., Ilhom Akobirshoev, Robert S. Dembo, and Monika Mitra. "Self-Injurious Behavior Among Adults With ASD: Hospitalizations, Length of Stay, and Costs of Resources to Deliver Care." *Psychiatric Services* 70, no. 6 (June 2019): 457–64. https://doi.org/10.1176/appi.ps.201800318.

Siegel, Matthew. "The Severe End of the Spectrum: Insights and Opportunities from the Autism Inpatient Collection (AIC)." *Journal of Autism and Developmental Disorders* 48, no. 11 (November 2018): 3641–46. https://doi.org/10.1007/s10803-018-3731-6.

Siegel, Matthew, Briana Milligan, Bruce Chemelski, David Payne, Beth Ellsworth, Jamie Harmon, Olivia Teer, and Kahsi A. Smith. "Specialized Inpatient Psychiatry for Serious Behavioral Disturbance in Autism and Intellectual Disability." *Journal of Autism and Developmental Disorders* 44, no. 12 (December 2014): 3026–32. https://doi.org/10.1007/s10803-014-2157-z.

Silverman, Jeremy M., Joseph D. Buxbaum, Nicolas Ramoz, James Schmeidler, Abraham Reichenberg, Eric Hollander, Gary Angelo, Christopher J. Smith, and Lauren A. Kryzak. "Autism-Related Routines and Rituals Associated with a Mitochondrial Aspartate/Glutamate Carrier SLC25A12 Polymorphism." *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* 147B, no. 3 (April 5, 2008): 408–10. https://doi.org/10.1002/ajmg.b.30614.

Silverman, Jeremy M., Christopher J. Smith, James Schmeidler, Eric Hollander, Brian A. Lawlor, Michael Fitzgerald, Joseph D. Buxbaum, Katherine Delaney, Patricia Galvin, and the Autism Genetic Research Exchange Consortium. "Symptom Domains in Autism and Related Conditions: Evidence for Familiality." *American Journal of Medical Genetics* 114, no. 1 (January 8, 2002): 64–73. https://doi.org/10.1002/ajmg.10048.

Silverman, Jill L., Audrey Thurm, Sarah B. Ethridge, Makayla M. Soller, Stela P. Petkova, Ted Abel, Melissa D. Bauman, et al. "Reconsidering Animal Models Used to Study Autism Spectrum Disorder: Current State and Optimizing Future." *Genes, Brain, and Behavior* 21, no. 5 (June 2022): e12803. https://doi.org/10.1111/gbb.12803.

Simon, Viktória, Pál Czobor, Sára Bálint, Ágnes Mészáros, and István Bitter. "Prevalence and Correlates of Adult Attention-Deficit Hyperactivity Disorder: Meta-Analysis." *British Journal of Psychiatry* 194, no. 3 (March 2009): 204–11. https://doi.org/10.1192/bjp.bp.107.048827.

Simonoff, Emily, Florence Mowlem, Oliver Pearson, Evdokia Anagnostou, Craig Donnelly, Eric Hollander, Bryan H. King, et al. "Citalopram Did Not Significantly Improve Anxiety in Children with Autism Spectrum Disorder Undergoing Treatment for Core Symptoms: Secondary Analysis of a Trial to Reduce Repetitive Behaviors." *Journal of Child and Adolescent Psychopharmacology* 32, no. 4 (May 1, 2022): 233–41. https://doi.org/10.1089/cap.2021.0137.

Simonoff, Emily, Andrew Pickles, Tony Charman, Susie Chandler, Tom Loucas, and Gillian Baird. "Psychiatric Disorders in Children With Autism Spectrum Disorders: Prevalence, Comorbidity, and Associated Factors in a Population-Derived Sample." *Journal of the American Academy of Child & Adolescent Psychiatry* 47, no. 8 (August 2008): 921–29. https://doi.org/10.1097/CHI.0b013e318179964f.

Singer, Harvey S., Christina Morris, Colin Gause, Matthew Pollard, Andrew W. Zimmerman, and Mikhail Pletnikov. "Prenatal Exposure to Antibodies from Mothers of Children with Autism Produces Neurobehavioral Alterations: A Pregnant Dam Mouse Model." *Journal of Neuroimmunology* 211, no. 1–2 (June 2009): 39–48. https://doi.org/10.1016/j.jneuroim.2009.03.011.

Siniscalco, Dario, James Jeffrey Bradstreet, Alessandra Cirillo, and Nicola Antonucci. "The in Vitro GcMAF Effects on Endocannabinoid System Transcriptionomics, Receptor Formation, and Cell Activity of Autism-Derived Macrophages." *Journal of Neuroinflammation* 11 (April 17, 2014): 78. https://doi.org/10.1186/1742-2094-11-78.

Siniscalco, Dario, James Jeffrey Bradstreet, Nataliia Sych, and Nicola Antonucci. "Perspectives on the Use of Stem Cells for Autism Treatment." *Stem Cells International* 2013 (2013): 1–7. https://doi.org/10.1155/2013/262438.

Siracusano, Martina, Valentina Postorino, Assia Riccioni, Leonardo Emberti Gialloreti, Monica Terribili, Paolo Curatolo, and Luigi Mazzone. "Sex Differences in Autism Spectrum Disorder: Repetitive Behaviors and Adaptive Functioning." *Children* 8, no. 5 (April 22, 2021): 325. https://doi.org/10.3390/children8050325.

Smith, Alan M., Marvin R. Natowicz, Daniel Braas, Michael A. Ludwig, Denise M. Ney, Elizabeth L. R. Donley, Robert E. Burrier, and David G. Amaral. "A Metabolomics Approach to Screening for Autism Risk in the Children's Autism Metabolome Project." *Autism Research* 13, no. 8 (August 2020): 1270–85. https://doi.org/10.1002/aur.2330.

Smith, Christopher J., Colleen M. Lang, Lauren Kryzak, Abraham Reichenberg, Eric Hollander, and Jeremy M. Silverman. "Familial Associations of Intense Preoccupations, an Empirical Factor of the Restricted, Repetitive Behaviors and Interests Domain of Autism." *Journal of Child Psychology and Psychiatry* 50, no. 8 (August 2009): 982–90. https://doi.org/10.1111/j.1469-7610.2009.02060.x.

Smith, Stephen E. P., Jennifer Li, Krassimira Garbett, Karoly Mirnics, and Paul H. Patterson. "Maternal Immune Activation Alters Fetal Brain Development through Interleukin-6." *The Journal of Neuroscience* 27, no. 40 (October 3, 2007): 10695–702. https://doi.org/10.1523/JNEUROSCI.2178-07.2007.

Society for Maternal-Fetal Medicine. "Prenatal Acetaminophen Use and Outcomes in Children." *American Journal of Obstetrics and Gynecology* 216, no. 3 (March 2017): B14–15. https://doi.org/10.1016/j.ajog.2017.01.021.

Sokolova, Elena, Anoek M. Oerlemans, Nanda N. Rommelse, Perry Groot, Catharina A. Hartman, Jeffrey C. Glennon, Tom Claassen, Tom Heskes, and Jan K. Buitelaar. "A Causal and Mediation Analysis of the Comorbidity Between Attention Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD)." *Journal of*

Autism and Developmental Disorders 47, no. 6 (2017): 1595–1604. https://doi.org/10.1007/s10803-017-3083-7.

Spanos, Marina, Tara Chandrasekhar, Soo-Jeong Kim, Robert M. Hamer, Bryan H. King, Christopher J. McDougle, Kevin B. Sanders, et al. "Rationale, Design, and Methods of the Autism Centers of Excellence (ACE) Network Study of Oxytocin in Autism to Improve Reciprocal Social Behaviors (SOARS-B)." *Contemporary Clinical Trials* 98 (November 2020): 106103. https://doi.org/10.1016/j.cct.2020.106103.

Spectrum | Autism Research News. "'Polygenic Risk Scores' for Autism, Explained," May 23, 2023. https://www.spectrumnews.org/news/polygenic-risk-scores-for-autism-explained/.

Spectrum | Autism Research News. "Rethinking Regression in Autism," August 2, 2017. https://www.spectrumnews.org/features/deep-dive/rethinking-regression-autism/.

Spectrum | Autism Research News. "The Multiple Hits Theory of Autism, Explained," May 1, 2019. https://www.spectrumnews.org/news/multiple-hits-theory-autism-explained/.

Spencer, Andrea E., Natalie Plasencia, Ying Sun, Cara Lucke, Haregnesh Haile, Rebecca Cronin, Stephen V. Faraone, Michael Jellinek, J. Michael Murphy, and Joseph Biederman. "Screening for Attention-Deficit/Hyperactivity Disorder and Comorbidities in a Diverse, Urban Primary Care Setting." *Clinical Pediatrics* 57, no. 12 (October 1, 2018): 1442–52. https://doi.org/10.1177/0009922818787329.

Spiers, Helen, Eilis Hannon, Leonard C. Schalkwyk, Rebecca Smith, Chloe C.Y. Wong, Michael C. O'Donovan, Nicholas J. Bray, and Jonathan Mill. "Methylomic Trajectories across Human Fetal Brain Development." *Genome Research* 25, no. 3 (March 2015): 338–52. https://doi.org/10.1101/gr.180273.114.

Spildrejorde, Mari, Athina Samara, Ankush Sharma, Magnus Leithaug, Martin Falck, Stefania Modafferi, Arvind Y. M. Sundaram, et al. "Multi-Omics Analysis of Paracetamol Exposure Identifies Dysregulated Genes Involved in Neurotoxicity and Neuronal Differentiation of Human Embryonic Stem Cells." Preprint. Cell Biology, December 10, 2022. https://doi.org/10.1101/2022.12.08.519620.

Stadnick, Nicole, Colby Chlebowski, Mary Baker-Ericzén, Margaret Dyson, Ann Garland, and Lauren Brookman-Frazee. "Psychiatric Comorbidity in Autism Spectrum Disorder: Correspondence between Mental Health Clinician Report and Structured Parent Interview." *Autism* 21, no. 7 (October 2017): 841–51. https://doi.org/10.1177/1362361316654083.

"State-Level Estimates of the Prevalence of Parent-Reported ADHD Diagnosis and Treatment Among U.S. Children and Adolescents, 2016 to 2019." Accessed June 6, 2023. https://doi.org/10.1177/10870547221099961.

Steiner, Michel A., and Carsten T. Wotjak. "Role of the Endocannabinoid System in Regulation of the Hypothalamic-Pituitary-Adrenocortical Axis." *Progress in Brain Research* 170 (2008): 397–432. https://doi.org/10.1016/S0079-6123(08)00433-0.

Stephens, David N., Hans S. Crombag, and Theodora Duka. "The Challenge of Studying Parallel Behaviors in Humans and Animal Models." *Current Topics in Behavioral Neurosciences* 13 (2013): 611–45. https://doi.org/10.1007/7854_2011_133.

Sterba, Sonya, Helen L. Egger, and Adrian Angold. "Diagnostic Specificity and Nonspecificity in the Dimensions of Preschool Psychopathology." *Journal of Child Psychology and Psychiatry* 48, no. 10 (October 2007): 1005–13. https://doi.org/10.1111/j.1469-7610.2007.01770.x.

Stergiakouli, Evie, Anita Thapar, and George Davey Smith. "Association of Acetaminophen Use During Pregnancy With Behavioral Problems in Childhood: Evidence Against Confounding." *JAMA Pediatrics* 170, no. 10 (October 1, 2016): 964. https://doi.org/10.1001/jamapediatrics.2016.1775.

Stern, Michael. "Insulin Signaling and Autism." *Frontiers in Endocrinology* 2 (October 14, 2011): 54. https://doi.org/10.3389/fendo.2011.00054.

Stevanovic, Dejan, Elisabet Wentz, Salmir Nasic, and Rajna Knez. "ASD with ADHD vs. ASD and ADHD Alone: A Study of the QbTest Performance and Single-Dose Methylphenidate Responding in Children and Adolescents." *BMC Psychiatry* 22, no. 1 (December 2022): 282. https://doi.org/10.1186/s12888-022-03878-3.

Storch, Eric A., Tanya K. Murphy, Gary R. Geffken, Daniel M. Bagner, Ohel Soto, Muhammad Sajid, Pam Allen, Erin M. Killiany, and Wayne K. Goodman. "Factor Analytic Study of the Children's Yale–Brown Obsessive–Compulsive Scale." *Journal of Clinical Child & Adolescent Psychology* 34, no. 2 (May 2005): 312–19. https://doi.org/10.1207/s15374424jccp3402_10.

Storch, Eric A., Steven A. Rasmussen, Lawrence H. Price, Michael J. Larson, Tanya K. Murphy, and Wayne K. Goodman. "Development and Psychometric Evaluation of the Yale–Brown Obsessive-Compulsive Scale—Second Edition." *Psychological Assessment* 22, no. 2 (2010): 223–32. https://doi.org/10.1037/a0018492.

Strang, John F., Laura G. Anthony, Benjamin E. Yerys, Kristina K. Hardy, Gregory L. Wallace, Anna C. Armour, Katerina Dudley, and Lauren Kenworthy. "The Flexibility Scale: Development and Preliminary Validation of a Cognitive Flexibility Measure in Children with Autism Spectrum Disorders." *Journal of Autism and Developmental Disorders* 47, no. 8 (August 2017): 2502–18. https://doi.org/10.1007/s10803-017-3152-y.

Strang, John F., Aron Janssen, Amy Tishelman, Scott F. Leibowitz, Lauren Kenworthy, Jenifer K. McGuire, Laura Edwards-Leeper, et al. "Revisiting the Link: Evidence of the Rates of Autism in Studies of Gender Diverse Individuals." *Journal of the American Academy of Child & Adolescent Psychiatry* 57, no. 11 (November 2018): 885–87. https://doi.org/10.1016/j.jaac.2018.04.023.

Streissguth, Ann Pytkowicz, Robert P. Treder, Helen M. Barr, Thomas H. Shepard, W. Archie Bleyer, Paul D. Sampson, and Donald C. Martin. "Aspirin and Acetaminophen Use by Pregnant Women and Subsequent Child IQ and Attention Decrements." *Teratology* 35, no. 2 (April 1, 1987): 211–19. https://doi.org/10.1002/tera.1420350207.

Subcommittee on Attention-Deficit/Hyperactivity Disorder, Steering Committee on Quality Improvement and Management. “ADHD: Clinical Practice Guideline for the Diagnosis, Evaluation, and Treatment of Attention-Deficit/Hyperactivity Disorder in Children and Adolescents.” *Pediatrics* 128, no. 5 (November 2011): 1007–22. https://doi.org/10.1542/peds.2011-2654.

Sudo, Nobuyuki, Yoichi Chida, Yuji Aiba, Junko Sonoda, Naomi Oyama, Xiao-Nian Yu, Chiharu Kubo, and Yasuhiro Koga. “Postnatal Microbial Colonization Programs the Hypothalamic-Pituitary-Adrenal System for Stress Response in Mice: Commensal Microbiota and Stress Response.” *The Journal of Physiology* 558, no. 1 (July 2004): 263–75. https://doi.org/10.1113/jphysiol.2004.063388.

Sullivan, Patrick F., Cecilia Magnusson, Abraham Reichenberg, Marcus Boman, Christina Dalman, Michael Davidson, Eyal Fruchter, et al. “Family History of Schizophrenia and Bipolar Disorder as Risk Factors for Autism.” *Archives of General Psychiatry* 69, no. 11 (November 1, 2012). https://doi.org/10.1001/archgenpsychiatry.2012.730.

Supekar, Kaustubh, Tara Iyer, and Vinod Menon. “The Influence of Sex and Age on Prevalence Rates of Comorbid Conditions in Autism: Influence of Sex & Age on Autism Comorbidities.” *Autism Research* 10, no. 5 (May 2017): 778–89. https://doi.org/10.1002/aur.1741.

Supekar, Kaustubh, and Vinod Menon. “Sex Differences in Structural Organization of Motor Systems and Their Dissociable Links with Repetitive/Restricted Behaviors in Children with Autism.” *Molecular Autism* 6, no. 1 (December 2015): 50. https://doi.org/10.1186/s13229-015-0042-z.

“Surveillance of ADHD Among Children in the United States: Validity and Reliability of Parent Report of Provider Diagnosis.” Accessed June 6, 2023. https://doi.org/10.1177/10870547221131979.

Suzuki, Katsuaki, Genichi Sugihara, Yasuomi Ouchi, Kazuhiko Nakamura, Masami Futatsubashi, Kiyokazu Takebayashi, Yujiro Yoshihara, et al. “Microglial Activation in Young Adults With Autism Spectrum Disorder.” *JAMA Psychiatry* 70, no. 1 (January 1, 2013): 49. https://doi.org/10.1001/jamapsychiatry.2013.272.

Swanson, J M, J A Sergeant, E Taylor, E J S Sonuga-Barke, P S Jensen, and D P Cantwell. “Attention-Deficit Hyperactivity Disorder and Hyperkinetic Disorder.” *THE LANCET* 351 (1998).

Sznajder, Kristin K., Douglas M. Teti, and Kristen H. Kjerulff. “Maternal Use of Acetaminophen during Pregnancy and Neurobehavioral Problems in Offspring at 3 Years: A Prospective Cohort Study.” Edited by Kang Sun. *PLOS ONE* 17, no. 9 (September 28, 2022): e0272593. https://doi.org/10.1371/journal.pone.0272593.

T Schultz, Stephen, and Georgianna G Gould. “Acetaminophen Use for Fever in Children Associated with Autism Spectrum Disorder.” *Autism-Open Access* 06, no. 02 (2016). https://doi.org/10.4172/2165-7890.1000170.

Takano, Tomoyuki. “Role of Microglia in Autism: Recent Advances.” *Developmental Neuroscience* 37, no. 3 (May 21, 2015): 195–202. https://doi.org/10.1159/000398791.

Tamiji, Javaneh, and Dorota A. Crawford. “The Neurobiology of Lipid Metabolism in Autism Spectrum Disorders.” *Neuro-Signals* 18, no. 2 (2010): 98–112. https://doi.org/10.1159/000323189.

Taylor, Bonnie P., Jianyou Liu, Wenzhu Mowrey, Eckhart Eule, Federico Bolognani, and Eric Hollander. “The Montefiore-Einstein Rigidity Scale-Revised (MERS-R): Development, Administration, Reliability, and Validity in Child and Adult Autism Spectrum Disorder (ASD).” *Journal of Psychiatric Research* 147 (March 2022): 142–47. https://doi.org/10.1016/j.jpsychires.2021.12.055.

Taylor, Briana J., Kevin B. Sanders, Marie Kyle, Kahsi A. Pedersen, Jeremy Veenstra-Vanderweele, and Matthew Siegel. “Inpatient Psychiatric Treatment of Serious Behavioral Problems in Children with Autism Spectrum Disorder (ASD): Specialized Versus General Inpatient Units.” *Journal of Autism and Developmental Disorders* 49, no. 3 (March 2019): 1242–49. https://doi.org/10.1007/s10803-018-3816-2.

Tebartz Van Elst, Ludger, Marion Pick, Monica Biscaldi, Thomas Fangmeier, and Andreas Riedel. “High-Functioning Autism Spectrum Disorder as a Basic Disorder in Adult Psychiatry and Psychotherapy: Psychopathological Presentation, Clinical Relevance and Therapeutic Concepts.” *European Archives of Psychiatry and Clinical Neuroscience* 263, no. S2 (November 2013): 189–96. https://doi.org/10.1007/s00406-013-0459-3.

Tetreault, Nicole A., Atiya Y. Hakeem, Sue Jiang, Brian A. Williams, Elizabeth Allman, Barbara J. Wold, and John M. Allman. “Microglia in the Cerebral Cortex in Autism.” *Journal of Autism and Developmental Disorders* 42, no. 12 (December 2012): 2569–84. https://doi.org/10.1007/s10803-012-1513-0.

Thapar, Anita, and Miriam Cooper. “Attention Deficit Hyperactivity Disorder.” *Lancet (London, England)* 387, no. 10024 (March 19, 2016): 1240–50. https://doi.org/10.1016/S0140-6736(15)00238-X.

Thapar, Anita, Miriam Cooper, Rachel Jefferies, and Evangelia Stergiakouli. “What Causes Attention Deficit Hyperactivity Disorder?” *Archives of Disease in Childhood* 97, no. 3 (March 2012): 260–65. https://doi.org/10.1136/archdischild-2011-300482.

The Autism and Developmental Disorders Inpatient Research Collaborative (ADDIRC), Benjamin L. Handen, Carla A. Mazefsky, Robin L. Gabriels, Kahsi A. Pedersen, Meredith Wallace, and Matthew Siegel. “Risk Factors for Self-Injurious Behavior in an Inpatient Psychiatric Sample of Children with Autism Spectrum Disorder: A Naturalistic Observation Study.” *Journal of Autism and Developmental Disorders* 48, no. 11 (November 2018): 3678–88. https://doi.org/10.1007/s10803-017-3460-2.

The Autism Genome Project Consortium. “Mapping Autism Risk Loci Using Genetic Linkage and Chromosomal Rearrangements.” *Nature Genetics* 39, no. 3 (March 2007): 319–28. https://doi.org/10.1038/ng1985.

The Diagnostic & Statistical Manual of Mental Disorders, 4th Edition

The Diagnostic & Statistical Manual of Mental Disorders, 4th Edition, Text Revision

The Diagnostic & Statistical Manual of Mental Disorders, Fifth Edition

"The CAST (Childhood Asperger Syndrome Test)." Accessed May 30, 2023. https://doi.org/10.1177/1362361302006001003.

The International ASEBA Consortium, Leslie A. Rescorla, Allison Adams, and Masha Y. Ivanova. "The CBCL/1½–5's DSM-ASD Scale: Confirmatory Factor Analyses Across 24 Societies." *Journal of Autism and Developmental Disorders* 50, no. 9 (September 2020): 3326–40. https://doi.org/10.1007/s10803-019-04189-5.

"The Prevalence of Attention Deficit/Hyperactivity Disorder Symptoms in Children and Adolescents With Autism Spectrum Disorder Without Intellectual Disability: A Systematic Review." Accessed June 9, 2023. https://doi.org/10.1177/10870547231177466.

"The Yale-Brown Obsessive Compulsive Scale: I. Development, Use, and Reliability," n.d.

"The Yale-Brown Obsessive Compulsive Scale: II. Validity," n.d.

Thiele, Kristin, Timo Kessler, Petra Arck, Annette Erhardt, and Gisa Tiegs. "Acetaminophen and Pregnancy: Short- and Long-Term Consequences for Mother and Child." *Journal of Reproductive Immunology* 97, no. 1 (March 2013): 128–39. https://doi.org/10.1016/j.jri.2012.10.014.

Thompson, John M. D., Karen E. Waldie, Clare R. Wall, Rinky Murphy, Edwin A. Mitchell, and the ABC study group. "Associations between Acetaminophen Use during Pregnancy and ADHD Symptoms Measured at Ages 7 and 11 Years." Edited by Kenji Hashimoto. *PLoS ONE* 9, no. 9 (September 24, 2014): e108210. https://doi.org/10.1371/journal.pone.0108210.

Thorsen, Morten, Niels Bilenberg, Lena Thorsen, and Tanja Maria Michel. "Oxidative Stress in Adults with Autism Spectrum Disorder: A Case Control Study." *Journal of Autism and Developmental Disorders* 52, no. 1 (January 2022): 275–82. https://doi.org/10.1007/s10803-021-04897-x.

Tick, Beata, Patrick Bolton, Francesca Happé, Michael Rutter, and Frühling Rijsdijk. "Heritability of Autism Spectrum Disorders: A Meta-Analysis of Twin Studies." *Journal of Child Psychology and Psychiatry* 57, no. 5 (May 2016): 585–95. https://doi.org/10.1111/jcpp.12499.

Tobiasova, Zuzana, Klaas H. B. Van Der Lingen, Lawrence Scahill, James F. Leckman, Yan Zhang, Wookjin Chae, James T. McCracken, et al. "Risperidone-Related Improvement of Irritability in Children with Autism Is Not Associated with Changes in Serum of Epidermal Growth Factor and Interleukin-13." *Journal of Child and Adolescent Psychopharmacology* 21, no. 6 (December 2011): 555–64. https://doi.org/10.1089/cap.2010.0134.

Toussaint, K., X. C. Yang, M. A. Zielinski, K. L. Reigle, S. D. Sacavage, S. Nagar, and R. B. Raffa. "What Do We (Not) Know about How Paracetamol (Acetaminophen) Works?" *Journal of Clinical Pharmacy and Therapeutics* 35, no. 6 (2010): 617–38. https://doi.org/10.1111/j.1365-2710.2009.01143.x.

Tovo-Rodrigues, Luciana, Bruna Celestino Schneider, Thais Martins-Silva, Bianca Del-Ponte, Christian Loret de Mola, Lavinia Schuler-Faccini, Fernanda Sales Luiz Vianna, et al. "Is Intrauterine Exposure to Acetaminophen Associated with Emotional and Hyperactivity Problems during Childhood? Findings from the 2004 Pelotas Birth Cohort." *BMC Psychiatry* 18, no. 1 (December 2018): 368. https://doi.org/10.1186/s12888-018-1942-1.

Towbin, Kenneth E., Anne Pradella, Tristan Gorrindo, Daniel S. Pine, and Ellen Leibenluft. "Autism Spectrum Traits in Children with Mood and Anxiety Disorders." *Journal of Child and Adolescent Psychopharmacology* 15, no. 3 (June 2005): 452–64. https://doi.org/10.1089/cap.2005.15.452.

Tromans, Sam, Guiqing Lily Yao, Reza Kiani, Regi Alexander, Mohammed Al-Uzri, and Traolach Brugha. "Study Protocol: An Investigation of the Prevalence of Autism among Adults Admitted to Acute Mental Health Wards: A Cross-Sectional Pilot Study." *BMJ Open* 9, no. 12 (December 2019): e033169. https://doi.org/10.1136/bmjopen-2019-033169.

Trønnes, Johanne N., Mollie Wood, Angela Lupattelli, Eivind Ystrom, and Hedvig Nordeng. "Prenatal Paracetamol Exposure and Neurodevelopmental Outcomes in Preschool-aged Children." *Paediatric and Perinatal Epidemiology* 34, no. 3 (May 2020): 247–56. https://doi.org/10.1111/ppe.12568.

Tubaro, Aurelia, Anna Giangaspero, Silvio Sosa, Roberto Negri, Gianpaolo Grassi, Salvatore Casano, Roberto Della Loggia, and Giovanni Appendino. "Comparative Topical Anti-Inflammatory Activity of Cannabinoids and Cannabivarins." *Fitoterapia* 81, no. 7 (October 2010): 816–19. https://doi.org/10.1016/j.fitote.2010.04.009.

Tureck, Kim, Johnny L. Matson, Paige Cervantes, and Nicole Turygin. "Autism Severity as a Predictor of Inattention and Impulsivity in Toddlers." *Developmental Neurorehabilitation* 18, no. 5 (September 3, 2015): 285–89. https://doi.org/10.3109/17518423.2013.807884.

Turner, Michelle. "Annotation: Repetitive Behaviour in Autism: A Review of Psychological Research." *Journal of Child Psychology and Psychiatry* 40, no. 6 (September 1999): 839–49. https://doi.org/10.1111/1469-7610.00502.

Tzavara, Eleni T., Dominic L. Li, Larissa Moutsimilli, Tiziana Bisogno, Vincenzo Di Marzo, Lee A. Phebus, George G. Nomikos, and Bruno Giros. "Endocannabinoids Activate Transient Receptor Potential Vanilloid 1 Receptors to Reduce Hyperdopaminergia-Related Hyperactivity: Therapeutic Implications." *Biological Psychiatry* 59, no. 6 (March 15, 2006): 508–15. https://doi.org/10.1016/j.biopsych.2005.08.019.

Uljarević, Mirko, Bronia Arnott, Sarah J. Carrington, Elizabeth Meins, Charles Fernyhough, Helen McConachie, Ann Le Couteur, and Susan R. Leekam. "Development of Restricted and Repetitive Behaviors from 15 to 77 Months: Stability of Two Distinct Subtypes?" *Developmental Psychology* 53, no. 10 (October 2017): 1859–68. https://doi.org/10.1037/dev0000324.

Uljarević, Mirko, Matthew N. Cooper, Keely Bebbington, Emma J. Glasson, Murray T. Maybery, Kandice Varcin, Gail A. Alvares, John Wray, Susan R. Leekam, and Andrew J.O. Whitehouse. "Deconstructing the Repetitive Behaviour Phenotype in Autism Spectrum Disorder through a Large Population-based Analysis." *Journal of Child Psychology and Psychiatry* 61, no. 9 (September 2020): 1030–42. https://doi.org/10.1111/jcpp.13203.

Uljarević, Mirko, and David W. Evans. "Relationship between Repetitive Behaviour and Fear across Normative Development, Autism Spectrum Disorder, and down Syndrome: Relationship between Insistence on Sameness and Fear." *Autism Research* 10, no. 3 (March 2017): 502–7. https://doi.org/10.1002/aur.1674.

Uljarević, Mirko, David W. Evans, Gail A. Alvares, and Andrew J. O. Whitehouse. "Short Report: Relationship between Restricted and Repetitive Behaviours in Children with Autism Spectrum Disorder and Their Parents." *Molecular Autism* 7, no. 1 (December 2016): 29. https://doi.org/10.1186/s13229-016-0091-y.

Uljarević, Mirko, Amanda L. Richdale, David W. Evans, Ru Ying Cai, and Susan R. Leekam. "Interrelationship between Insistence on Sameness, Effortful Control and Anxiety in Adolescents and Young Adults with Autism Spectrum Disorder (ASD)." *Molecular Autism* 8, no. 1 (December 2017): 36. https://doi.org/10.1186/s13229-017-0158-4.

Umbricht, Daniel, Marta Del Valle Rubido, Eric Hollander, James T McCracken, Frederick Shic, Lawrence Scahill, Jana Noeldeke, et al. "A Single Dose, Randomized, Controlled Proof-Of-Mechanism Study of a Novel Vasopressin 1a Receptor Antagonist (RG7713) in High-Functioning Adults with Autism Spectrum Disorder." *Neuropsychopharmacology* 42, no. 9 (August 2017): 1914–23. https://doi.org/10.1038/npp.2016.232.

Upadhya, Sudarshan C., Padmashree S. Tirumalai, Michael R. Boyd, Toshiyuki Mori, and Vijayalakshmi Ravindranath. "Cytochrome P4502E (CYP2E) in Brain: Constitutive Expression, Induction by Ethanol and Localization by Fluorescence in Situ Hybridization." *Archives of Biochemistry and Biophysics* 373, no. 1 (January 2000): 23–34. https://doi.org/10.1006/abbi.1999.1477.

US EPA, OA. "Health - Neurodevelopmental Disorders." Overviews and Factsheets, May 29, 2015. https://www.epa.gov/americaschildrenenvironment/health-neurodevelopmental-disorders.

"Use of the Autism Diagnostic Observation Schedule (ADOS) in a Clinical Setting." Accessed May 14, 2023. https://doi.org/10.1177/1362361310379241.

"Using Horn's Parallel Analysis Method in Exploratory Factor Analysis for Determining the Number of Factors." *Educational Sciences: Theory & Practice*, 2016. https://doi.org/10.12738/estp.2016.2.0328.

Uzunova, Genoveva, Stefano Pallanti, and Eric Hollander. "Excitatory/Inhibitory Imbalance in Autism Spectrum Disorders: Implications for Interventions and Therapeutics." *The World Journal of Biological Psychiatry* 17, no. 3 (April 2, 2016): 174–86. https://doi.org/10.3109/15622975.2015.1085597.

Van Der Miesen, Anna I.R., Hannah Hurley, and Annelou L.C. De Vries. "Gender Dysphoria and Autism Spectrum Disorder: A Narrative Review." *International Review of Psychiatry* 28, no. 1 (January 2, 2016): 70–80. https://doi.org/10.3109/09540261.2015.1111199.

Van Meter, Anna R., Margaret H. Sibley, Pankhuree Vandana, Boris Birmaher, Mary A. Fristad, Sarah Horwitz, Eric A. Youngstrom, Robert L. Findling, and L. Eugene Arnold. "The Stability and Persistence of Symptoms in Childhood-Onset ADHD." *European Child & Adolescent Psychiatry*, June 4, 2023. https://doi.org/10.1007/s00787-023-02235-3.

Van Orden, Kimberly A., Tracy K. Witte, Kelly C. Cukrowicz, Scott R. Braithwaite, Edward A. Selby, and Thomas E. Joiner. "The Interpersonal Theory of Suicide." *Psychological Review* 117, no. 2 (2010): 575–600. https://doi.org/10.1037/a0018697.

Vandewouw, Marlee M., Jessica Brian, Jennifer Crosbie, Russell J. Schachar, Alana Iaboni, Stelios Georgiades, Robert Nicolson, et al. "Identifying Replicable Subgroups in Neurodevelopmental Conditions Using Resting-State Functional Magnetic Resonance Imaging Data." *JAMA Network Open* 6, no. 3 (March 13, 2023): e232066. https://doi.org/10.1001/jamanetworkopen.2023.2066.

Varela Casal, Paloma, Flavia Lorena Esposito, Imanol Morata Martínez, Alba Capdevila, Maria Solé Puig, Núria de la Osa, Lourdes Ezpeleta, et al. "Clinical Validation of Eye Vergence as an Objective Marker for Diagnosis of ADHD in Children." *Journal of Attention Disorders* 23, no. 6 (April 1, 2019): 599–614. https://doi.org/10.1177/1087054717749931.

Vargas, Diana L., Caterina Nascimbene, Chitra Krishnan, Andrew W. Zimmerman, and Carlos A. Pardo. "Neuroglial Activation and Neuroinflammation in the Brain of Patients with Autism." *Annals of Neurology* 57, no. 1 (2005): 67–81. https://doi.org/10.1002/ana.20315.

Vasudeva, Sara B., and Eric Hollander. "Body Dysmorphic Disorder in Patients With Autism Spectrum Disorder: A Reflection of Increased Local Processing and Self-Focus." *American Journal of Psychiatry* 174, no. 4 (April 2017): 313–16. https://doi.org/10.1176/appi.ajp.2016.16050559.

Vegni, Nicoletta, Caterina D'Ardia, and Giulia Torregiani. "Empathy, Mentalization, and Theory of Mind in Borderline Personality Disorder: Possible Overlap With Autism Spectrum Disorders." *Frontiers in Psychology* 12 (February 11, 2021): 626353. https://doi.org/10.3389/fpsyg.2021.626353.

Velie, E. M., G. Block, G. M. Shaw, S. J. Samuels, D. M. Schaffer, and M. Kulldorff. "Maternal Supplemental and Dietary Zinc Intake and the Occurrence of Neural Tube Defects in California." *American Journal of Epidemiology* 150, no. 6 (September 15, 1999): 605–16. https://doi.org/10.1093/oxfordjournals.aje.a010059.

Ventola, Pamela E., Daniel Yang, Sebiha M. Abdullahi, Courtney A. Paisley, Megan L. Braconnier, and Denis G. Sukhodolsky. "Brief Report: Reduced Restricted and Repetitive Behaviors after Pivotal Response Treatment." *Journal of Autism and Developmental Disorders* 46, no. 8 (August 2016): 2813–20. https://doi.org/10.1007/s10803-016-2813-6.

Verheij, C., A. Louwerse, J. Van Der Ende, M. L. J. M. Eussen, A. R. Van Gool, F. Verheij, F. C. Verhulst, and K. Greaves-Lord. "The Stability of Comorbid Psychiatric Disorders: A 7 Year Follow Up of Children with Pervasive Developmental Disorder-Not Otherwise Specified." *Journal of Autism and Developmental Disorders* 45, no. 12 (December 2015): 3939–48. https://doi.org/10.1007/s10803-015-2592-5.

Viberg, Henrik, Per Eriksson, Torsten Gordh, and Anders Fredriksson. "Paracetamol (Acetaminophen) Administration during Neonatal Brain Development Affects Cognitive Function and Alters Its Analgesic and Anxiolytic Response in Adult Male Mice." *Toxicological Sciences: An Official Journal of the Society of Toxicology* 138, no. 1 (March 2014): 139–47. https://doi.org/10.1093/toxsci/kft329.

Visser, Susanna N., Melissa L. Danielson, Mark L. Wolraich, Michael H. Fox, Scott D. Grosse, Linda A. Valle, Joseph R. Holbrook, Angelika H. Claussen, and Georgina Peacock. "Vital Signs: National and State-Specific Patterns of Attention Deficit/Hyperactivity Disorder Treatment Among Insured Children Aged 2–5 Years — United States, 2008–2014." *MMWR. Morbidity and Mortality Weekly Report* 65, no. 17 (May 6, 2016): 443–50. https://doi.org/10.15585/mmwr.mm6517e1.

Vlenterie, Richelle, Mollie E. Wood, Ragnhild Eek Brandlistuen, Nel Roeleveld, Marleen M.H.J. van Gelder, and Hedvig Nordeng. "Neurodevelopmental Problems at 18 Months among Children Exposed to Paracetamol *in Utero* : A Propensity Score Matched Cohort Study." *International Journal of Epidemiology* 45, no. 6 (August 31, 2016): dyw192. https://doi.org/10.1093/ije/dyw192.

Volkmar, Fred, Matthew Siegel, Marc Woodbury-Smith, Bryan King, James McCracken, and Matthew State. "Practice Parameter for the Assessment and Treatment of Children and Adolescents With Autism Spectrum Disorder." *Journal of the American Academy of Child & Adolescent Psychiatry* 53, no. 2 (February 2014): 237–57. https://doi.org/10.1016/j.jaac.2013.10.013.

Vuillermot, Stéphanie, Eliza Joodmardi, Thomas Perlmann, Sven Ove Ögren, Joram Feldon, and Urs Meyer. "Prenatal Immune Activation Interacts with Genetic Nurr1 Deficiency in the Development of Attentional Impairments." *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience* 32, no. 2 (January 11, 2012): 436–51. https://doi.org/10.1523/JNEUROSCI.4831-11.2012.

Wammes, Linda J, Harriet Mpairwe, Alison M Elliott, and Maria Yazdanbakhsh. "Helminth Therapy or Elimination: Epidemiological, Immunological, and Clinical Considerations." *The Lancet Infectious Diseases* 14, no. 11 (November 2014): 1150–62. https://doi.org/10.1016/S1473-3099(14)70771-6.

Wan, Hongquan, Chunguo Zhang, He Li, Shuxin Luan, and Chang Liu. "Association of Maternal Diabetes with Autism Spectrum Disorders in Offspring." *Medicine* 97, no. 2 (January 12, 2018): e9438. https://doi.org/10.1097/MD.0000000000009438.

Wang, Chengzhong, Hua Geng, Weidong Liu, and Guiqin Zhang. "Prenatal, Perinatal, and Postnatal Factors Associated with Autism." *Medicine* 96, no. 18 (May 5, 2017): e6696. https://doi.org/10.1097/MD.0000000000006696.

Wang, Lv, Claus T Christophersen, Michael J Sorich, Jacobus P Gerber, Manya T Angley, and Michael A Conlon. “Increased Abundance of Sutterella Spp. and Ruminococcus Torques in Feces of Children with Autism Spectrum Disorder.” *Molecular Autism* 4, no. 1 (December 2013): 42. https://doi.org/10.1186/2040-2392-4-42.

Wang, Ying, Shiming Tang, Shunsheng Xu, Shenhong Weng, and Zhongchun Liu. “Maternal Body Mass Index and Risk of Autism Spectrum Disorders in Offspring: A Meta-Analysis.” *Scientific Reports* 6 (September 30, 2016): 34248. https://doi.org/10.1038/srep34248.

Warrier, Varun, Vivienne Chee, Paula Smith, Bhismadev Chakrabarti, and Simon Baron-Cohen. “A Comprehensive Meta-Analysis of Common Genetic Variants in Autism Spectrum Conditions.” *Molecular Autism* 6, no. 1 (December 2015): 49. https://doi.org/10.1186/s13229-015-0041-0.

Wasserman, Stacey, Rupa Iyengar, William F. Chaplin, Dryden Watner, Shulamit E. Waldoks, Evdokia Anagnostou, Latha Soorya, and Eric Hollander. “Levetiracetam versus Placebo in Childhood and Adolescent Autism: A Double-Blind Placebo-Controlled Study:” *International Clinical Psychopharmacology* 21, no. 6 (November 2006): 363–67. https://doi.org/10.1097/01.yic.0000224787.13782.0f.

Watanabe, Takamitsu, Rebecca P. Lawson, Ylva S.E. Walldén, and Geraint Rees. “A Neuroanatomical Substrate Linking Perceptual Stability to Cognitive Rigidity in Autism.” *The Journal of Neuroscience* 39, no. 33 (August 14, 2019): 6540–54. https://doi.org/10.1523/JNEUROSCI.2831-18.2019.

Wei, H., I. Alberts, and X. Li. “Brain IL-6 and Autism.” *Neuroscience* 252 (November 2013): 320–25. https://doi.org/10.1016/j.neuroscience.2013.08.025.

Weigand, U. W., R. C. Chou, D. Maulik, and G. Levy. “Assessment of Biotransformation during Transfer of Propoxyphene and Acetaminophen across the Isolated Perfused Human Placenta.” *Pediatric Pharmacology (New York, N.Y.)* 4, no. 3 (1984): 145–53.

Weiss, M., D. Worling, and M. Wasdell. “A Chart Review Study of the Inattentive and Combined Types of ADHD.” *Journal of Attention Disorders* 7, no. 1 (September 2003): 1–9. https://doi.org/10.1177/108705470300700101.

“What Does It Mean to Be Transdiagnostic and How Would We Know? | American Journal of Psychiatry.” Accessed May 11, 2023. https://ajp.psychiatryonline.org/doi/10.1176/appi.ajp.2020.20030243.

White, Susan W., Grace Lee Simmons, Katherine O. Gotham, Caitlin M. Conner, Isaac C. Smith, Kelly B. Beck, and Carla A. Mazefsky. “Psychosocial Treatments Targeting Anxiety and Depression in Adolescents and Adults with Autism Spectrum Disorder: Review of the Latest Research and Recommended Future Directions.” *Current Psychiatry Reports* 20, no. 10 (August 28, 2018): 82. https://doi.org/10.1007/s11920-018-0949-0.

Wilkes, B.J., and M.H. Lewis. “The Neural Circuitry of Restricted Repetitive Behavior: Magnetic Resonance Imaging in Neurodevelopmental Disorders and Animal Models.” *Neuroscience & Biobehavioral Reviews* 92 (September 2018): 152–71. https://doi.org/10.1016/j.neubiorev.2018.05.022.

Willcutt, Erik G. "The Prevalence of DSM-IV Attention-Deficit/Hyperactivity Disorder: A Meta-Analytic Review." *Neurotherapeutics* 9, no. 3 (July 2012): 490–99. https://doi.org/10.1007/s13311-012-0135-8.

Williams, Brent L., Mady Hornig, Timothy Buie, Margaret L. Bauman, Myunghee Cho Paik, Ivan Wick, Ashlee Bennett, Omar Jabado, David L. Hirschberg, and W. Ian Lipkin. "Impaired Carbohydrate Digestion and Transport and Mucosal Dysbiosis in the Intestines of Children with Autism and Gastrointestinal Disturbances." Edited by Steven Jacobson. *PLoS ONE* 6, no. 9 (September 16, 2011): e24585. https://doi.org/10.1371/journal.pone.0024585.

Williams, Brent L., Mady Hornig, Tanmay Parekh, and W. Ian Lipkin. "Application of Novel PCR-Based Methods for Detection, Quantitation, and Phylogenetic Characterization of *Sutterella* Species in Intestinal Biopsy Samples from Children with Autism and Gastrointestinal Disturbances." Edited by Christine Biron. *MBio* 3, no. 1 (March 2012): e00261-11. https://doi.org/10.1128/mBio.00261-11.

Williams, Jo, Carrie Allison, Fiona Scott, Carol Stott, Patrick Bolton, Simon Baron-Cohen, and Carol Brayne. "The Childhood Asperger Syndrome Test (CAST): Test–Retest Reliability." *Autism* 10, no. 4 (July 2006): 415–27. https://doi.org/10.1177/1362361306066612.

Williams, Jo, Fiona Scott, Carol Stott, Carrie Allison, Patrick Bolton, Simon Baron-Cohen, and Carol Brayne. "The CAST (Childhood Asperger Syndrome Test): Test Accuracy." *Autism* 9, no. 1 (February 2005): 45–68. https://doi.org/10.1177/1362361305049029.

Williams, Joanna G., Carrie Allison, Fiona J. Scott, Patrick F. Bolton, Simon Baron-Cohen, Fiona E. Matthews, and Carol Brayne. "The Childhood Autism Spectrum Test (CAST): Sex Differences." *Journal of Autism and Developmental Disorders* 38, no. 9 (October 2008): 1731–39. https://doi.org/10.1007/s10803-008-0558-6.

Williams, Ozge C., Sakshi Prasad, Amanda McCrary, Erica Jordan, Vishi Sachdeva, Sheryl Deva, Harendra Kumar, Jayati Mehta, Purushottam Neupane, and Aditi Gupta. "Adult Attention Deficit Hyperactivity Disorder: A Comprehensive Review." *Annals of Medicine and Surgery* 85, no. 5 (April 12, 2023): 1802–10. https://doi.org/10.1097/MS9.0000000000000631.

Williams, Tanishia A., Audrey E. Mars, Steven G. Buyske, Edward S. Stenroos, Rong Wang, Marivic F. Factura-Santiago, George H. Lambert, and William G. Johnson. "Risk of Autistic Disorder in Affected Offspring of Mothers with a Glutathione S-Transferase P1 Haplotype." *Archives of Pediatrics & Adolescent Medicine* 161, no. 4 (April 2007): 356–61. https://doi.org/10.1001/archpedi.161.4.356.

Wilson, Rachel I., and Roger A. Nicoll. "Endocannabinoid Signaling in the Brain." *Science (New York, N.Y.)* 296, no. 5568 (April 26, 2002): 678–82. https://doi.org/10.1126/science.1063545.

Wolf, Michael S., Jennifer King, Kara Jacobson, Lorenzo Di Francesco, Stacy Cooper Bailey, Rebecca Mullen, Danielle McCarthy, Marina Serper, Terry C. Davis, and Ruth M. Parker. "Risk of Unintentional Overdose with Non-Prescription Acetaminophen

Products.” *Journal of General Internal Medicine* 27, no. 12 (December 2012): 1587–93. https://doi.org/10.1007/s11606-012-2096-3.

Wolraich, Mark L, Laura McGuinn, and Melissa Doffing. “Treatment of Attention Deficit Hyperactivity Disorder in Children and Adolescents: Safety Considerations.” *Drug Safety* 30, no. 1 (2007): 17–26. https://doi.org/10.2165/00002018-200730010-00003.

Wong, Cheryl M., and Eric Hollander. “New Dimensions in the OCD Spectrum: Autism, Pathologic Gambling, and Compulsive Buying.” *CNS Spectrums* 1, no. 2 (November 1996): 44–53. https://doi.org/10.1017/S1092852900002649.

Wong, Christine T., Isabel Bestard-Lorigados, and Dorota A. Crawford. “Autism-Related Behaviors in the Cyclooxygenase-2-Deficient Mouse Model.” *Genes, Brain, and Behavior* 18, no. 1 (January 2019): e12506. https://doi.org/10.1111/gbb.12506.

Wood, Mollie E. “Associations between Prenatal Acetaminophen Exposure and Child Neurodevelopment: Truth, Bias, or a Bit of Both?” *Paediatric and Perinatal Epidemiology* 34, no. 3 (May 2020): 233–36. https://doi.org/10.1111/ppe.12658.

Wood, N. S., N. Marlow, K. Costeloe, A. T. Gibson, and A. R. Wilkinson. “Neurologic and Developmental Disability after Extremely Preterm Birth. EPICure Study Group.” *The New England Journal of Medicine* 343, no. 6 (August 10, 2000): 378–84. https://doi.org/10.1056/NEJM200008103430601.

Wu, Ka, Chao Guo, Xiuli Lu, Xinmou Wu, Hongmei Pan, and Min Su. “Impact of Perinatal Exposure to Acetaminophen on Hepatocellular Metabolic Function in Offspring.” *American Journal of Translational Research* 8, no. 12 (December 15, 2016): 5646–52.

Wu, Long-Jun, Beth Stevens, Shumin Duan, and Brian A. MacVicar. “Microglia in Neuronal Circuits.” *Neural Plasticity* 2013 (2013): 1–3. https://doi.org/10.1155/2013/586426.

Wu, S., F. Wu, Y. Ding, J. Hou, J. Bi, and Z. Zhang. “Advanced Parental Age and Autism Risk in Children: A Systematic Review and Meta-Analysis.” *Acta Psychiatrica Scandinavica* 135, no. 1 (January 2017): 29–41. https://doi.org/10.1111/acps.12666.

Wu, Wei-Li, Catherine E. Adams, Karen E. Stevens, Ke-Huan Chow, Robert Freedman, and Paul H. Patterson. “The Interaction between Maternal Immune Activation and Alpha 7 Nicotinic Acetylcholine Receptor in Regulating Behaviors in the Offspring.” *Brain, Behavior, and Immunity* 46 (May 2015): 192–202. https://doi.org/10.1016/j.bbi.2015.02.005.

Xu, Guifeng, Jin Jing, Katherine Bowers, Buyun Liu, and Wei Bao. “Maternal Diabetes and the Risk of Autism Spectrum Disorders in the Offspring: A Systematic Review and Meta-Analysis.” *Journal of Autism and Developmental Disorders* 44, no. 4 (April 2014): 766–75. https://doi.org/10.1007/s10803-013-1928-2.

Yoshimasu, Kouichi, Chikako Kiyohara, Shigeki Takemura, and Kunihiko Nakai. “A Meta-Analysis of the Evidence on the Impact of Prenatal and Early Infancy Exposures to Mercury on Autism and Attention Deficit/Hyperactivity Disorder in the Childhood.” *Neurotoxicology* 44 (September 2014): 121–31. https://doi.org/10.1016/j.neuro.2014.06.007.

Young, Susan, and Kelly Cocallis. “A Systematic Review of the Relationship Between Neurodiversity and Psychosexual Functioning in Individuals with Autism Spectrum Disorder (ASD) or Attention-Deficit/Hyperactivity Disorder (ADHD).” *Neuropsychiatric Disease and Treatment* Volume 19 (June 2023): 1379–95. https://doi.org/10.2147/NDT.S319980.

Young, Susan, Jack Hollingdale, Michael Absoud, Patrick Bolton, Polly Branney, William Colley, Emily Craze, et al. “Guidance for Identification and Treatment of Individuals with Attention Deficit/Hyperactivity Disorder and Autism Spectrum Disorder Based upon Expert Consensus.” *BMC Medicine* 18 (May 25, 2020): 146. https://doi.org/10.1186/s12916-020-01585-y.

Ystrom, Eivind, Kristin Gustavson, Ragnhild Eek Brandlistuen, Gun Peggy Knudsen, Per Magnus, Ezra Susser, George Davey Smith, and Camilla Stoltenberg. “Prenatal Exposure to Acetaminophen and Risk of ADHD.” *Pediatrics* 140, no. 5 (2017): 9. https://doi.org/10.1542/peds.2016-3840.

Yu, Yanjie, Ashmita Chaulagain, Sindre Andre Pedersen, Stian Lydersen, Bennett L. Leventhal, Peter Szatmari, Branko Aleksic, Norio Ozaki, and Norbert Skokauskas. “Pharmacotherapy of Restricted/Repetitive Behavior in Autism Spectrum Disorder:A Systematic Review and Meta-Analysis.” *BMC Psychiatry* 20, no. 1 (December 2020): 121. https://doi.org/10.1186/s12888-020-2477-9.

Yui, Kunio, George Imataka, Yohei Kawasak, and Hiroshi Yamada. “Increased ω-3 Polyunsaturated Fatty Acid/Arachidonic Acid Ratios and Upregulation of Signaling Mediator in Individuals with Autism Spectrum Disorders.” *Life Sciences* 145 (January 2016): 205–12. https://doi.org/10.1016/j.lfs.2015.12.039.

Zablotsky, Benjamin, Matthew D. Bramlett, and Stephen J. Blumberg. “The Co-Occurrence of Autism Spectrum Disorder in Children with ADHD.” *Journal of Attention Disorders* 24, no. 1 (January 2020): 94–103. https://doi.org/10.1177/1087054717713638.

Zenclussen, Ana Claudia. “CD4+CD25+ T Regulatory Cells in Murine Pregnancy.” *Journal of Reproductive Immunology* 65, no. 2 (April 2005): 101–10. https://doi.org/10.1016/j.jri.2005.01.003.

Zerbo, Ousseny, Ana-Maria Iosif, Cheryl Walker, Sally Ozonoff, Robin L. Hansen, and Irva Hertz-Picciotto. “Is Maternal Influenza or Fever During Pregnancy Associated with Autism or Developmental Delays? Results from the CHARGE (CHildhood Autism Risks from Genetics and Environment) Study.” *Journal of Autism and Developmental Disorders* 43, no. 1 (January 2013): 25–33. https://doi.org/10.1007/s10803-012-1540-x.

Zerbo, Ousseny, Cathleen Yoshida, Judith K Grether, Judy Van De Water, Paul Ashwood, Gerald N Delorenze, Robin L Hansen, Marty Kharrazi, and Lisa A Croen. “Neonatal Cytokines and Chemokines and Risk of Autism Spectrum Disorder: The Early Markers for Autism (EMA) Study: A Case-Control Study.” *Journal of Neuroinflammation* 11, no. 1 (2014): 113. https://doi.org/10.1186/1742-2094-11-113.

Zhao, Lizi, and Gisèle Pickering. "Paracetamol Metabolism and Related Genetic Differences." *Drug Metabolism Reviews* 43, no. 1 (February 2011): 41–52. https://doi.org/10.3109/03602532.2010.527984.

Zheng, Shuting, Ryan Adams, Julie Lounds Taylor, Florencia Pezzimenti, and Somer L Bishop. "Depression in Independent Young Adults on the Autism Spectrum: Demographic Characteristics, Service Use, and Barriers." *Autism* 25, no. 7 (October 2021): 1960–72. https://doi.org/10.1177/13623613211008276.

Zhong, Caichen, Jillian Tessing, Brian K. Lee, and Kristen Lyall. "Maternal Dietary Factors and the Risk of Autism Spectrum Disorders: A Systematic Review of Existing Evidence." *Autism Research: Official Journal of the International Society for Autism Research* 13, no. 10 (October 2020): 1634–58. https://doi.org/10.1002/aur.2402.

Zhou, Melissa S., Madeeha Nasir, Luis C. Farhat, Minjee Kook, Bekir B. Artukoglu, and Michael H. Bloch. "Meta-Analysis: Pharmacologic Treatment of Restricted and Repetitive Behaviors in Autism Spectrum Disorders." *Journal of the American Academy of Child & Adolescent Psychiatry* 60, no. 1 (January 2021): 35–45. https://doi.org/10.1016/j.jaac.2020.03.007.

Zilkha, N., Y. Kuperman, and T. Kimchi. "High-Fat Diet Exacerbates Cognitive Rigidity and Social Deficiency in the BTBR Mouse Model of Autism." *Neuroscience* 345 (March 2017): 142–54. https://doi.org/10.1016/j.neuroscience.2016.01.070.

Zou, Mingyang, Yu Liu, Shu Xie, Luxi Wang, Dexin Li, Ling Li, Feng Wang, et al. "Alterations of the Endocannabinoid System and Its Therapeutic Potential in Autism Spectrum Disorder." *Open Biology* 11, no. 2 (January 4, 2021): 200306. https://doi.org/10.1098/rsob.200306.

Zwick, Georg Peter. "Neuropsychological Assessment in Autism Spectrum Disorder and Related Conditions." *Dialogues in Clinical Neuroscience* 19, no. 4 (December 31, 2017): 373–79. https://doi.org/10.31887/DCNS.2017.19.4/gzwick.